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13. ABSTRACT *(Maximum 200 words)*

Individuals exercise and work in a wide range of environmental conditions (temperature, humidity, sun, wind, rain, other water). Depending upon the environmental conditions, metabolic rate, and clothing, exercise. can accentuate either heat gain or heat loss, causing body temperature to rise or fall. Humans normally regulate body (core) temperatures near 37°C, and fluctuations within the narrow range of 35° to 41 °C can degrade exercise performance. Fluctuations outside that range can be lethal (1). Therefore, heat or cold stress can have profound effects on exercise capability as well as morbidity and mortality. In this chapter the term exercise refers to dynamic exercise, and training refers to repeated days of exercise in a specific modality. Throughout this chapter, stress refers to environmental exercise conditions tending to influence the body's heat content and strain refers to physiological consequences of stress. The magnitude of stress and the resulting strain depends upon the complex interaction of environmental factors (e.g., ambient conditions, clothing), the individual's biological characteristics (e.g., acclimatization status, body size) and exercise task (e.g., metabolic rate, duration). Acclimatization refers to adaptations to both natural (acclimatization) and artificial (acclimation) environmental conditions. This chapter examines the effects of both heat stress and cold stress on physiological responses and exercise capabilities. Human thermoregulation during exercise is addressed, but more detailed reviews on human thermoregulation during environmental stress can be found elsewhere (2-4). This chapter includes information on pathogenesis of exertional heat illness and exertional hypothermia, since exercise can increase morbidity and mortality from thermal injury. Other chapters emphasize acute and chronic (training) exercise, whereas in this chapter the focus is on acute and chronic (acclimatization) environmental exposure, although the effects of exercise training on physiological responses during thermal (heat or cold) stress are discussed.

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CHAPTER 26

Physiological Systems and Their Responses to Conditions of Heat and Cold

MICHAEL N. SAWKA AND ANDREW J. YOUNG

Introduction

Individuals exercise and work in a wide range of environmental conditions (temperature, humidity, sun, wind, rain, other water). Depending upon the environmental conditions, metabolic rate, and clothing, exercise can accentuate either heat gain or heat loss, causing body temperature to rise or fall. Humans normally regulate body (core) temperatures near 37°C, and fluctuations within the narrow range of 35° to 41°C can degrade exercise performance. Fluctuations outside that range can be lethal (1). Therefore, heat or cold stress can have profound effects on exercise capability as well as morbidity and mortality.

In this chapter the term *exercise* refers to dynamic exercise, and *training* refers to repeated days of exercise in a specific modality. Throughout this chapter, *stress* refers to environmental exercise conditions tending to influence the body's heat content and *strain* refers to physiological consequences of stress. The magnitude of stress and the resulting strain depends upon the complex interaction of environmental factors (e.g., ambient conditions, clothing), the individual's biological characteristics (e.g., acclimatization status, body size) and exercise task (e.g., metabolic rate, duration). Acclimatization refers to adaptations to both natural (acclimatization) and artificial (acclimation) environmental conditions.

This chapter examines the effects of both heat stress and cold stress on physiological responses and exercise capabilities. Human thermoregulation during exercise is addressed, but more detailed reviews on human thermoregulation during environmental stress can be found elsewhere (2–4). This chapter includes information on pathogenesis of exertional heat illness and exertional hypothermia, since exercise can increase morbidity and mortality from thermal injury. Other chapters emphasize acute and chronic (training) exercise, whereas in this chapter the focus is on acute and chronic (acclimatization) environmental exposure, although the effects of exercise training on physiological responses during thermal (heat or cold) stress are discussed.

Thermal Balance and Control

Biophysics of Heat Exchange and Balance

Figure 26.1 schematically shows energy (heat) transfers of an exercising athlete. Muscular contraction produces metabolic heat that is transferred from the active muscle to blood and the body core. Since skeletal muscle contraction is about 20% efficient, about 80% of expended energy is released as heat and must be dissipated from the body to avoid heat storage and increasing body temperature. Physiological adjustments redirect blood flow from the body core to periphery, thereby facilitating heat transfer from within the body to the skin, where it can be dissipated into the environment. Heat exchange between skin and the environment is governed

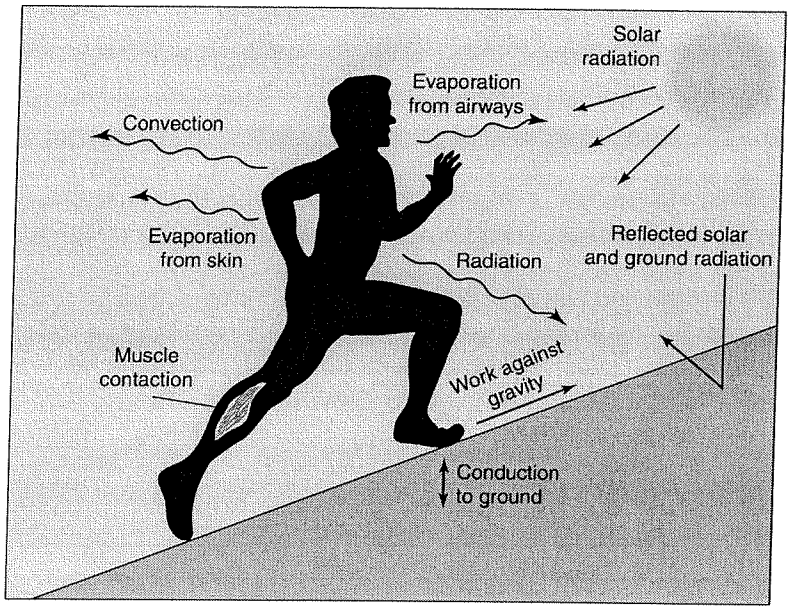

FIGURE 26.1 Avenues of energy (heat) exchange for an athlete performing exercise in air.

by biophysical properties dictated by surrounding air or water temperature; air humidity; air or water motion; solar, sky, and ground radiation; and clothing (5). The biophysical avenues of this heat exchange are conduction, convection, radiation, and evaporation. The nonevaporative (conduction, convection, and radiation) avenues are often collectively called dry heat exchange.

Conduction is heat transfer between two solid objects in direct contact, and convection is heat exchange between a surface and a fluid, including air or water. Heat exchange by conduction or convection occurs as long as there is a temperature gradient between the body surface and contacting object or surrounding fluid. When a person is standing, walking, or running and wearing shoes, heat exchange by conduction is minimal, because the thermal gradients between the body and contacted solids are usually small. Convective heat exchange is facilitated if the surrounding fluid is moving (e.g., wind, water circulation), relative to the body surface. In air environments, convective heat transfer can be significantly increased by wind if clothing does not create a barrier, and for swimmers convective heat loss can be very large, even when the difference between body surface and surrounding fluid temperature is small, since water's heat capacity is much greater than that of air: its convective heat transfer coefficient is about 25 times that of air (3). Heat loss by convection to air or water occurs when the air or water temperature is below body temperature; conversely, heat gain by convection from air or water occurs when the temperature exceeds that of the body.

Heat loss by radiation occurs when surrounding sun, sky, ground, or other large natural or manmade objects have lower surface temperatures than the body, and heat gain by radiation occurs when surrounding objects have higher surface temperatures than body surface temperature. Radiative heat exchange is independent of air temperature or motion. Accordingly, temperature combinations of the sky, ground, and surrounding objects may result in body heat gain due to radiation, even though the air temperature is below that of the body. For example, on a very sunny day a mountaineer on a snowy surface may gain a significant amount of heat despite low air temperature, and heat loss from exposed skin is greater under a clear night sky than in daylight, even when ambient air temperatures are the same.

Evaporative heat loss occurs with the phase change when liquid turns to water vapor. For humans, physiological vectors of evaporative heat loss are associated with breathing and perspiration. When water secreted onto the skin via sweat glands or rained or splashed onto the skin evaporates, or water from respiratory passages evaporates, the kinetic energy of the motion of the water molecule, latent heat of evaporation, eliminates heat from the body. Evaporative cooling accounts for almost all heat loss during exercise at ambient temperatures equal to or above skin temperatures.

Body temperature reflects the balance between internal heat production and body heat transfer to the environment. The energy balance equation describes these relationships between the body and environment:

$$S = M - (\pm W) \pm (R + C) \pm K - E$$

where S = rate of body heat storage; M = rate of metabolic energy (heat) production; W = mechanical work of either concentric (positive) or eccentric (negative) exercise; R + C =

rate of radiant and convective energy exchanges; K = rate of conduction (important only during direct contact with an object, such as clothing, or a substance, such as water); and E = rate of evaporative loss. The sum of these, heat storage, represents heat gain if positive or heat loss if negative. Body temperature increases when S is positive, decreases when S is negative, and remains constant when S equals zero (5).

Physiological Thermoregulation

Humans regulate core temperature through two collaborative processes: behavioral and physiological temperature regulation (2). Behavioral temperature regulation operates through conscious alterations in behavior that influence heat storage. It includes things like modifying activity levels, changing clothes, and seeking shade or shelter. Physiological temperature regulation operates through responses that are independent of conscious voluntary behavior. It includes control of: (*a*) rate of metabolic heat production (e.g., shivering), (*b*) body heat distribution via the blood from the core to the skin (e.g., cutaneous vasodilation and constriction), and (*c*) sweating. Persons often choose to ignore effective behavioral thermoregulation strategies because of their motivation to win or complete a task.

The function of the human thermoregulatory system is shown schematically in Figure 26.2 (2). This scheme presumes that thermal receptors in the core and skin send information about their temperature to some central integrator. Any deviation between the controlled variable (body temperature) and a reference variable (e.g., set-point temperature) constitutes a load error that generates a thermal command signal to control sweating, vasodilation, vasoconstriction, and shivering. The notion of such a thermal command signal is supported by two observations: (*a*) Core temperature (70–90%) and skin temperature (10–30%) provide similar influence on the control of both sweating and skin blood flow responses. (*b*) The threshold temperatures for both sweating and skin blood flow are simultaneously shifted by a similar magnitude, by factors such as biological rhythms, endogenous pyrogens, and heat acclimatization. It is useful to think of such similar and simultaneous shifts in various thermoregulatory thresholds as representing (or as being the result of) a shift in thermoregulatory set point (6). In contrast, dynamic exercise can increase the threshold temperature for skin blood flow but not alter the threshold for sweating, so this would not be interpreted as a change in set-point temperature (7).

A disturbance in the regulated variable, core temperature, elicits graded heat loss or heat gain responses. Peripheral (skin) and central (brain, spinal column, large vessels) thermal receptors provide afferent input into hypothalamic thermoregulatory centers (8), where this information is processed, producing a load error and proportionate thermoregulatory command signal to initiate responses to regain and maintain heat balance. In addition, very small changes in hypothalamus (anterior preoptic area) temperature elicits changes in the thermoregulatory effector responses, as this area contains many neurons that increase their firing rate in response either to warming or to cooling. The magnitudes of changes in heat loss (e.g., sweating, skin blood flow), heat

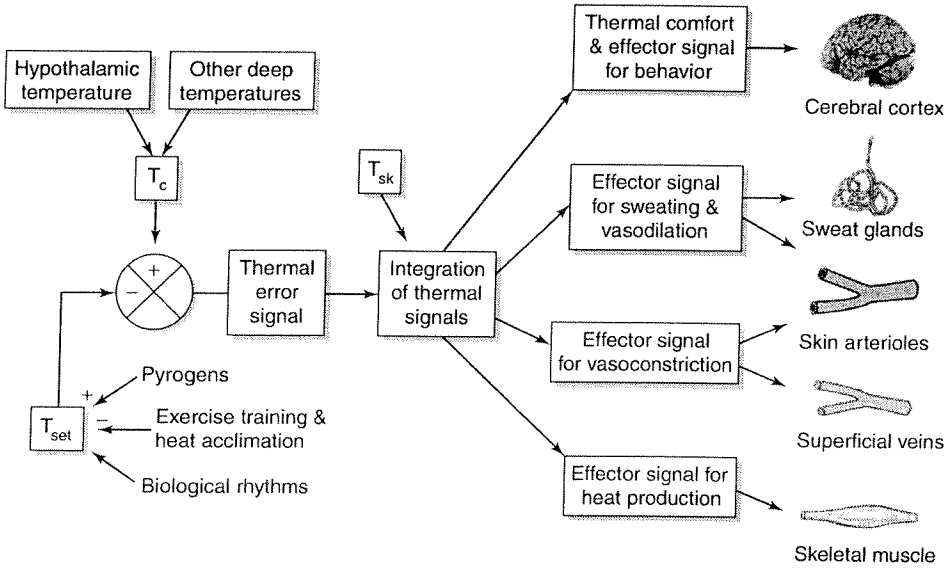

FIGURE 26.2 Human thermoregulation. *(Adapted from Sawka MN, Wenger CB, Pandolf KB. Thermoregulatory responses to acute exercise heat stress and heat acclimation. In Fregly MJ, Blatteis CM, eds. Handbook of Physiology: Section 4. Environmental Physiology. New York: Oxford University, 1996;160.)*

conservation (e.g., vasoconstriction) or production (e.g., shivering) are proportional to the displacement of the regulated variable (core temperature) from the thermoregulatory set-point temperature. The set-point temperature serves as a reference (analogous to a thermostat setting) in the control of all of the thermoregulatory responses (9). There is controversy as to whether a set point truly exists, and there are alternative theories (10).

Exercise and fever can both increase core temperature, but acute exercise does not involve a set-point increase, whereas fever does (6). Figure 26.3 shows schematically the difference in thermoregulatory control between fever and exercise hyperthermia (11). Metabolic heat production increases immediately with the initiation of exercise, causing heat storage and thus a load error (–e) or difference between set-point temperature and the elevated core temperature. The set-point temperature is unchanged, and therefore heat-dissipating responses are elicited as core temperature increases until heat loss responses sufficient to match heat production are achieved and a new thermal balance is established. When exercise stops, heat loss exceeds heat production, so core temperature falls back toward the set point. This diminishes the signal (load error) eliciting the heat dissipation responses, and they decline to baseline levels as the thermal balance conditions prevailing before exercise are reestablished. Therefore, the primary event elevating core temperature during exercise is increased metabolic heat production.

In fever, the primary event is an elevation of set-point temperature, which initially causes a negative load error (11). Heat-dissipating responses are inhibited and/or heat production is stimulated until core temperature increases enough to correct the load error and reestablish thermal balance at a new set-point temperature in which heat production and heat loss are near their values before the fever. The inhibition of heat dissipation and/or stimulation of heat production acts independently as a result of the person's thermal state and environmental temperature. When the fever abates (set point returns to normal), the heat-dissipating responses are increased and/or heat production is reduced until normal thermal balance is reestablished. If individuals perform exercise while having a fever, their exercise hyperthermia is imposed above the fever temperature.

In summary, when changes in metabolic heat production or environmental temperature upsets the thermal balance between heat dissipation and heat production, heat will be stored or lost from the body, and temperatures in the core or skin or both will change. Those temperature changes will be detected by the thermal receptors. In response to information from these receptors, the thermoregulatory controller in the central nervous system will call for responses that alter heat loss and/or production. Unless the thermal stress exceeds the capacity of the thermoregulatory system, these responses will continue until they are sufficient to restore heat balance and prevent further change in body temperatures.

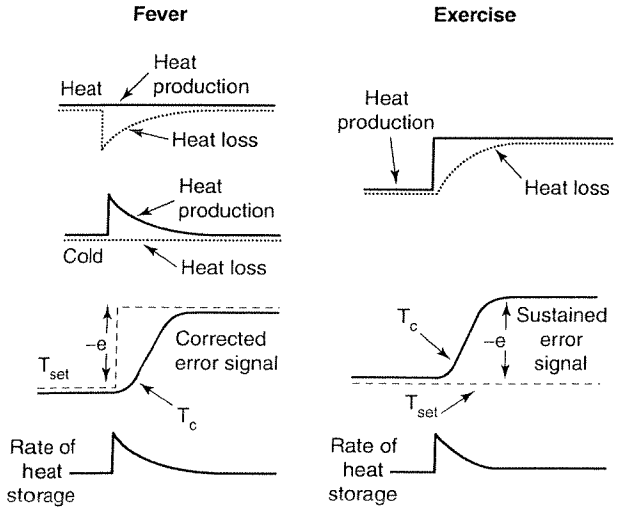

FIGURE 26.3 Differences between the elevation of core temperature in fever and during exercise. *(Adapted from Stitt JT. Fever versus hyperthermia. Fed Proc 1979;38:41, 42.)*

Core Temperature

Fundamental to the experimental study of human temperature regulation is the measurement of body core temperature. Core temperature is measured either to provide an estimate of the core temperature input to thermoregulatory control or to estimate average internal temperature to compute changes in heat storage in the core (2). Brain (i.e., hypothalamic) temperature during exercise is probably similar to blood temperature; however, recent evidence suggests that it could be slightly higher (12). There is no one true core temperature because temperature varies among sites deep inside the body. The temperature within a given deep body region depends upon (*a*) the local metabolic rate of the surrounding tissues, (*b*) the source and magnitude of local blood flow, and (*c*) the temperature gradients between contiguous body regions. Considerable temperature gradients exist between and within orifices, body cavities, and blood vessels. For resting humans, internal organs and viscera within the body core produce about 70% of the metabolic heat. During dynamic exercise, however, skeletal muscles produce up to about 90% of the metabolic heat. Because metabolic heat sources change during exercise as compared to rest, temperature changes measured in one body region during exercise may be disproportionate to changes measured in other body regions. For example, during rest in a comfortable environment, skeletal muscle temperature is lower than core temperature, but during exercise, the temperature within active skeletal muscle often exceeds core temperature (temperature within inactive skeletal muscle usually does not increase). Blood perfusing active skeletal muscles is warmed, and the blood carries that heat to

other body regions, and consequently, core temperature is elevated.

Core temperature during exercise is often measured at the esophagus, rectum, mouth, tympanum, and auditory meatus (2). Measurement methods employed for each of these sites and the advantages and disadvantages of each are summarized in Table 26.1. In brief, most thermal physiologists consider esophageal temperature to be the most accurate and reliable noninvasive index of core temperature for humans, followed in preference by rectal temperature and gastrointestinal tract temperature measured using ingestible temperature sensor pills. The pills are ideally suited for ambulatory monitoring outside of laboratories. Tympanic and auditory meatus temperatures are widely used, but all are influenced to some degree by head and face skin temperatures and ambient temperature and sensitive to inaccuracies related to proper placement of the sensor. Rectal and gastrointestinal tract temperatures are often slightly higher and slower to respond than esophageal temperature.

Heat Stress

Core Temperature Response to Exercise

Figure 26.4 illustrates the core (rectal and esophageal) temperature responses to two exercise bouts interspersed by a brief rest period (13). During exercise and recovery both measures of core temperature show similar patterns but with somewhat different kinetics and absolute values (rectal temperature slower to respond and slightly higher). Heat production increases almost immediately at the onset of exercise, so that during the early stages of exercise, rate of heat production exceeds rate of dissipation, and the undissipated heat is stored, primarily in the core, causing core temperature to rise. As core temperature rises, heat-dissipating reflexes are elicited, and the rate of heat storage decreases, so that core temperature rises more slowly. Eventually, as exercise continues, heat dissipation increases sufficiently to balance heat production, and essentially steady-state values are achieved. When exercise is discontinued, core temperature returns toward baseline levels, and with subsequent exercise the process is repeated.

The heat exchange components to achieve steady-state core temperature are dependent upon the environmental conditions; however, exercise type (arm vs. leg) can influence regional body heat exchange (14). Figure 26.5 illustrates the whole-body heat exchanges that might be expected during exercise (650-W metabolic rate) in a broad range of ambient temperatures (5–36°C, dry-bulb temperatures with low humidity) (15). The difference between metabolic rate and total heat loss represents the energy used for mechanical work (and heat storage or heat debt, if none steady-state). Total heat loss, and therefore heat storage and elevation of core temperature, are essentially the same in all environments. The relative contribution of dry and evaporative heat exchange to the total heat loss, however, varies with the environment. As the ambient temperature increases, the gradient for dry heat exchange diminishes and evaporative heat exchange becomes more important. When ambient temperature approaches or exceeds skin temperature, evaporative heat exchange will account for virtually all heat loss.

Heat stress can be divided into compensated heat stress (CHS) and uncompensated heat stress (UCHS). CHS and UCHS are primarily determined by biophysical factors (environment, clothing, metabolic rate) and are modestly affected by biological status (heat acclimatization and hydration status). CHS exists when heat loss occurs at a rate in balance with heat production, so that a steady-state core temperature can be achieved at a sustainable level for a requisite activity. CHS occurs in most situations. UCHS occurs when the individual's evaporative cooling requirements exceed the environment's evaporative cooling capacity. During UCHS, an individual cannot achieve steady-state core temperature, and core temperature rises until exhaustion occurs at physiological limits. Examples of UCHS include performing intense exercise in oppressive heat and wearing a football uniform while exercising in hot weather (16).

During CHS and dynamic exercise, the magnitude of the steady-state core temperature elevation is largely independent of the environment and is proportional to the metabolic rate. This idea, that steady-state core temperature elevation during exercise is independent of the CHS environment, may be inconsistent with the personal experience of some individuals. This is because there are biophysical limits to heat exchange between the environment and the

TABLE 26.1 Core Temperature Measurements

Site	Advantage	Disadvantage
Esophageal	Accurate, rapid response	Uncomfortable, affected by swallowing
Rectal	Accurate, easy to measure	Slow response, cultural objections
Auditory canal, tympanic membrane	Easy to measure	Inaccurate (biased by skin and ambient temperature), uncomfortable
Oral	Easy to measure	Inaccurate (affected by mouth breathing)
Pill	Accurate, easy to measure	Pill movement from stomach and location influence measurement

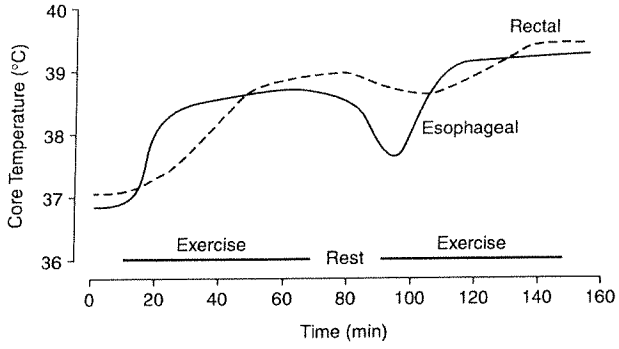

FIGURE 26.4 Core temperature (rectal and esophageal) responses to two exercise bouts separated by a brief rest period. *(Reprinted from Sawka MN, Wenger CB. Physiological responses to acute exercise heat stress. In KB Pandolf, Sawka MN, Gonzalez RR, eds. Human Performance Physiology and Environmental Medicine at Terrestrial Extremes. Indianapolis: Cooper, 1988;110.)*

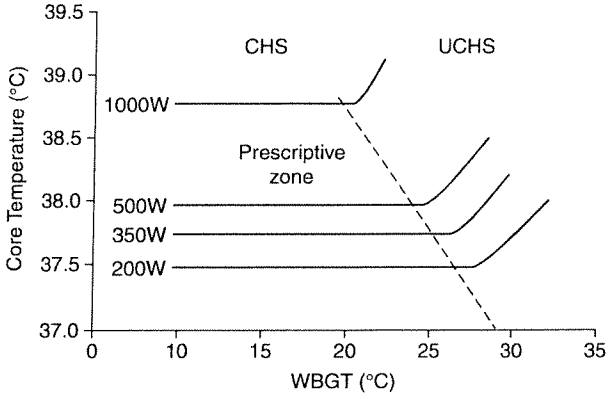

FIGURE 26.6 Possible core temperature (steady-state) responses during exercise at four metabolic rates during compensable (CHS) and uncompensable (UCHS) heat stress. *(Adapted from Lind AR. A physiological criterion for setting thermal environmental limits for everyday work. J Appl Physiol 1963;18:53.)*

performer, and core temperature may have to be elevated further to facilitate heat exchange. Actually, the magnitude of core temperature increase during exercise is independent of the environment only within a range of CHS conditions, or the "prescriptive zone" (17). Outside the prescriptive zone within CHS, a steady-state core temperature will be achieved, but it is elevated. Figure 26.6 provides an illustration of the steady-state core temperature elevation for a lightly clothed person that might be expected at several metabolic rates and wet-bulb globe temperature (WBGT) conditions in low humidity (17). The 200-, 350-, and 500-W metabolic rates approximate very light, moderate, and hard-intensity dynamic exercise for occupational tasks, respectively. For many individuals, the upper limit for sustained exercise corresponds to a metabolic rate of about 1000 W. The prescriptive zone narrows as metabolic rate increases.

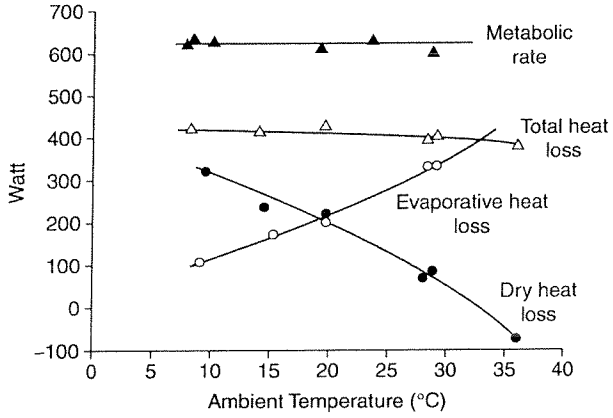

FIGURE 26.5 Heat exchange during exercise in a broad range of environmental temperatures with low humidity. *(From Nielsen M. Die Regulation der Körpertemperatur bei Muskelarbeit. Scand Arch Physiol 1938;9:216.)*

During CHS the core temperature increases in proportion to the metabolic rate during exercise (17,18). The greater the metabolic rate, the higher the steady-state core temperature during exercise. This relationship between metabolic rate and core temperature holds true for a given person but not always for comparisons between people. The use of relative intensity (percent of maximal oxygen uptake) rather than absolute metabolic rate (absolute intensity) reduces the variability between subjects for the core temperature elevation during exercise in CHS.

Acute Exercise Heat Stress

Sweating and Evaporative Cooling

For humans, unlike most animals, respiratory evaporative cooling is small compared to total skin evaporative cooling. Human skin provides the advantage over respiratory tract by virtue of a greater surface area directly exposed to ambient conditions for evaporation. Thermoregulatory sweating can begin within a few minutes after starting muscular exercise. The onset time of thermoregulatory sweating can vary and is influenced by skin temperature, acclimatization status, hydration status, and nonthermal stimuli (19).

The increase in thermoregulatory sweating closely parallels the increase in body temperature. As the sweating rate increases, first sweat glands are recruited and then sweat secretion per gland increases. Therefore, the sweat secretion for a given region of skin is dependent upon both the density of sweat glands and secretion per gland (20). In addition, different body regions of skin have different sweating responses (threshold and/or sensitivity) for a given core temperature. For a given core temperature, the back and chest have the highest sweating rates, while the limbs have relatively high sweating rates only after a substantial elevation in core temperature (21).

The eccrine glands secrete sweat onto the skin, which causes evaporative cooling by conversion of sweat from liquid to water vapor. The evaporation rate is dependent upon the gradient between the skin and water vapor pressure and the coefficient of evaporative heat transfer; and the wider the water vapor gradient, the greater the rate of evaporation for a given mass transfer coefficient (5). When 1 g of sweat is vaporized at 30°C, 2.43 kJ of heat energy becomes kinetic energy (latent heat of evaporation). The following calculations provide the minimal sweat requirements for persons performing exercise at 600-W metabolic rate in severe heat, in which only evaporative cooling is possible. If the activity is 20% efficient, the remaining 80% of metabolic energy produced is converted to heat in the body, so that 480 W (0.48 $kJ \cdot s^{-1}$, or 28.8 $kJ \cdot min^{-1}$) must be dissipated to avoid heat storage. The specific heat of body tissue (amount of energy required for 1 g of tissue to increase temperature by 1°C) approximates 3.5 $kJ \cdot °C^{-1}$, so a 70-kg man has a heat capacity of 245 $kJ \cdot °C^{-1}$. If this person performed exercise in a hot environment that enabled only evaporative heat loss and did not sweat, body temperature would increase by approximately 1.0°C every 8.5 min (245 kJ per °C, 28.8 $kJ \cdot min^{-1}$). Since the latent heat of evaporation is 2.43 $kJ \cdot g^{-1}$, this person would need to evaporate approximately 12 g of sweat (28.8 $kJ \cdot min^{-1}$/2.43 $kJ \cdot g^{-1}$) per minute, or 0.72 $L \cdot h^{-1}$. Sweating, however, is not 100% efficient, because secreted sweat can drip from the body and not be evaporated. Therefore, higher sweat secretions than calculated may actually be needed to satisfy demands for cooling.

Skin Blood Flow and Dry Heat Loss

Blood transfers heat by convection from the deep body tissues to the skin. When core and skin temperatures are low enough that sweating does not occur, raising skin blood flow brings skin temperature nearer to blood temperature, and lowering skin blood flow brings skin temperature nearer to ambient temperature. Thus, the body can control dry heat loss by varying skin blood flow and thereby skin temperature. When sweating occurs, the tendency of skin blood flow to warm the skin is approximately balanced by the tendency of sweat evaporation to cool the skin. Therefore, after sweating has begun, skin blood flow serves primarily to deliver to the skin the heat that is being removed by sweat evaporation. Skin blood flow and sweating thus work in tandem to dissipate heat.

Skin circulation is affected by temperature in two ways: local skin temperature affects the vascular smooth muscle directly, and temperatures of the core and of the skin elsewhere on the body affect skin blood flow by reflexes operating through the sympathetic nervous system. Blood flow in much of the human skin is under dual vasomotor control (22). In the palm of the hand, sole of the foot, lips, ears, and nose, adrenergic vasoconstrictor fibers are probably the predominant vasomotor innervation, and the vasodilation that occurs in these regions during heat exposure is largely the result of withdrawing vasoconstrictor activity. Over most of the skin area, there is minimal vasoconstrictor activity at skin temperatures of about 39°C and above (23), and vasodilation during heat exposure depends on intact sympathetic innervation. This vasodilation depends on the action of neural signals and as such is sometimes referred to as active vasodilation. Both active vasoconstriction and active vasodilation play a major part in controlling skin blood flow of the arm, thigh, and calf. However, active vasodilation is primarily responsible for controlling skin blood flow on the torso. The mechanism or mechanisms responsible for active cutaneous vasodilation are not fully understood. It is known that active cutaneous vasodilation is usually closely associated with sweating. Whether this link is due to a cotransmitter or linkage to sudomotor neural activity is unresolved, but possible vasoactive substances include vasoactive intestinal peptide, calcitonin gene–related peptide, substance P, and nitric oxide (22).

Skin blood flow requirements to achieve heat dissipation needs change with the skin (ambient conditions and sweat evaporation) and core temperatures. Table 26.2 provides calculations for minimal skin blood flow requirements at several core and skin temperature levels. These estimates assume that blood entering and leaving the cutaneous vasculature is equal to core and skin temperatures, respectively (24). If the exercise-mediated heat production is 10 $kcal \cdot min^{-1}$ (about 698 W of heat production so requires a metabolic rate of 872 W [about 2.5 L oxygen uptake] with 80% of energy converted to heat) and core temperature is 38°C, the minimal skin blood flow requirement will be about 5 $L \cdot min^{-1}$ if skin temperature is 36°C but will decrease to 2.5 $L \cdot min^{-1}$ if skin temperature decreases to 34°C. This clearly demonstrates the advantage of improved evaporative cooling (lowering skin temperature) on reducing cardiovascular requirements for skin blood flow.

TABLE 26.2 Skin Blood Flow Requirements for Several Core and Skin Temperatures During Exercise Heat Stress

Heat Production	Core Temperature	Skin Temperature	Core-to-Skin Gradient	Skin Blood Flow
10	38	36	2	5.0
10	38	34	4	2.5
10	39	36	3	3.3

Heat production in kilocalories per minute; temperatures in degrees Celsius; blood flow in liters per minute.
SKBF = 1/SH · Hp/Tc – Tsk. SKBF = skin blood flow; SH = specific heat of blood ~1 Kcal per °C per liter of blood; HP = heat production.

During exercise under environmental conditions that do not allow skin cooling, the body can employ a different approach to reduce skin blood flow requirements, one of which is to tolerate an additional elevation in core temperature. If skin temperature remains at 36°C, a core temperature elevation by 1°C increases the core-to-skin temperature gradient and minimal skin blood flow requirement from 5.0 to 3.3 L · minute^{-1} (Table 26.2). This explains the advantage of an elevated core temperature outside the prescriptive zone during CHS (Fig. 26.6). Therefore, accentuated core temperature elevations during exercise should not always be interpreted as an undesirable outcome; they may reflect a strategy to reduce cardiovascular strain to sustain thermal balance and exercise performance.

Cardiovascular Effects

During exercise heat stress, maintaining high skin blood flow can impose a substantial burden on the cardiovascular system (24). Skin blood flow can approach 8 L · min^{-1} for an average adult during heat stress. High skin blood flow is associated with reduced right atrial pressure and filling (24). This reduction in cardiac filling occurs because the cutaneous venous bed is large and compliant and dilates during heat stress. For these reasons, the venous bed of the skin—especially below heart level—tends to become engorged with blood at the expense of central blood volume as skin blood flow increases. Sweat secretion can result in a net body water loss, thereby reducing blood volume. Therefore, heat stress can reduce cardiac filling both through pooling of blood in the skin and through reduced blood volume. To compensation for reduced cardiac filling during rest and exercise, cardiac contractility increases to maintain stroke volume because of elevated sympathetic activity and parasympathetic withdrawal and perhaps temperature effects on the cardiac pacemaker cells. In addition, whole-body heating in resting subjects can reduce carotid baroreflex control of blood pressure and vagal baroreflex regulation of heart rate, which might contribute to orthostatic intolerance during heat stress (25,26).

During exercise in the heat, the primary cardiovascular challenge is to increase cardiac output sufficiently to support both high skin blood flow for heat dissipation and high muscle blood flow for metabolism. Exercise heat stress increases skin blood flow and usually sustains muscle blood flow relative to temperate conditions; however, reduced muscle blood flow has been observed during maximal exercise with heat stress (27). Figure 26.7 provides an analysis of cardiac output and distribution during rest and dynamic exercise in temperate and hot climates (24). This figure depicts cardiac output as being elevated during mild and perhaps moderate exercise in the heat compared to similar exercise in temperate conditions. During high- and maximal-intensity exercise in the heat, cardiac output can be below levels observed during similar exercise in temperate conditions. The lower cardiac output during maximal exercise results from an inability to maintain stroke volume because of skin blood flow and pooling (28).

Brain, spinal cord, and coronary blood flow are believed to be unaffected by exercise heat stress. However, as a result of increased sympathetic activity and thermal receptor stimulation, visceral (splanchnic, renal) blood flow is reduced by both exercise and heat stress. The visceral blood flow reductions are graded to the intensity of exercise, and the effects of exercise and heat seem to be additive (24). The mechanisms responsible for changes in this splanchnic blood flow are probably quite complex (29). Reduced visceral blood flow allows a corresponding diversion of cardiac output to skin and

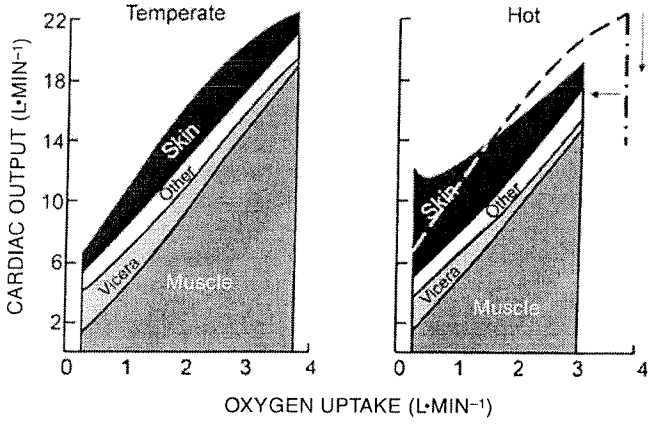

FIGURE 26.7 Comparison of cardiac output responses to exercise performed at various metabolic rates in temperate and heat stress conditions. *(From Rowell LB. Human circulation: regulation during physical stress. New York: Oxford University Press, 1986:385.)*

about 39–40°C despite large differences in exercise duration. Likewise, time to fatigue in a cool (10°C) environment even after completing a muscle glycogen–reducing regimen is even markedly longer (>30 min) than time to fatigue for glycogen-loaded subjects in a hot (30°C) environment (38). Finally, muscle heating does not have a consistent effect on maximal voluntary contraction (MVC) and will generally reduce muscle endurance at a given percentage of MVC.

Heat Strain Mechanisms of Exhaustion

Table 26.3 presents physiological mechanisms that can reduce exercise performance in the heat. These mechanisms include increased thermal and cardiovascular strain, diminished central nervous system drive for exercise, accelerated glycogen depletion with increased metabolite accumulation, and perhaps increased discomfort. The exact mechanism or mechanisms are unknown but probably depend upon the specific heat stress, exercise task, and biomedical state of the individual. For example, the increased glycogen depletion would be critical to reducing performance only if the individual could minimize the adverse consequences of elevated cardiovascular strain.

Core temperature provides the most reliable physiological index to predict the incidence of exhaustion from heat strain (39). However, as discussed earlier, the skin temperature associated with a given core temperature can greatly modify the physiological strain. Figure 26.9 illustrates some relationships between core temperature and incidence of exhaustion from heat strain for heat-acclimated persons exercising in uncompensable (most likely very hot skin) and compensable (most likely cool skin) heat stress (40). During uncompensable heat stress, exhaustion is rarely associated with a core temperature below 38°C, and exhaustion will almost always occur before a core temperature of 40°C (39). Remember, a skin temperature above 35°C, as seen during uncompensable heat stress, will be associated with greater cardiovascular strain for a given core temperature (41) and therefore result in earlier exhaustion. If skin is relatively cool, higher core temperatures can be better sustained during exercise.

Some elite athletes can tolerate core temperatures above 40°C and continue to exercise. It is unknown whether their tolerance to high core temperatures results from extensive heat acclimatization, training practices, natural selection, or a combination of these factors. Regardless, for a given athlete the exercise heat stress condition and how the high core temperature with skin temperature is achieved contribute to this variability. For example, skin temperature that starts low and rises throughout exercise will allow a higher core temperature than if skin temperature is constantly hot. For many hot weather conditions, the expected incidence of exhaustion will probably fall somewhere between the two extremes in Figure 26.9.

The role of cardiovascular strain on reducing exercise performance has been discussed earlier. Another possible mechanism is that hyperthermia itself might be an impetus for a central nervous system–mediated diminished drive to exercise (42). There is growing evidence in both humans and animals of a role for serotonin contributing to the genesis of fatigue from exercise hyperthermia. The effects of serotonin in the nervous system may be excitatory or inhibitory, depending largely on the brain region and the receptor involved, but general agreement is that acute serotonin accumulation produces an overall feeling of sleepiness and lethargy. Peripheral measurements of prolactin concentrations have been used as a biomarker for brain serotonergic activity and may increase with core temperature elevations above 38°C (43). Therefore, heat strain may elevate brain serotonin levels and reduce the desire to continue exercise. Consistent with this hypothesis, exercise in the heat can induce changes in electroencephalographic activity from the frontal cortex that are consistent with changes seen with drowsiness (44).

The first convincing demonstration that the central nervous system may mediate reduced exercise performance in the heat was provided by Lars Nybo and Bodil Nielsen of Denmark (42) (Milestone of Discovery). These experiments demonstrated that exercise hyperthermia (40°C) markedly reduces voluntary force-generating capacity during a prolonged MVC in both active (leg) and inactive (forearm) muscles when compared to that measured at a core temperature of 38°C. However, electrical stimulation of these hyperthermic muscles restored total force generation to that of the lower-temperature trial. These data strongly suggest a central

TABLE 26.3 Physiological Mechanisms Contributing to Reduced Exercise Performance During Heat Stress

Mechanism	Causes	Consequence
Cardiovascular strain	High skin blood flow Compliant skin Dehydration	Inability to maintain required cardiac output and blood pressure
Central fatigue	High brain temperature	Reduced neural drive to exercise
Thermal discomfort	Hot and wet skin High heart rate	Reduced desire to exercise
Muscle glycogen depletion	High muscle temperature High sympathetic activity	Insufficient carbohydrate substrate

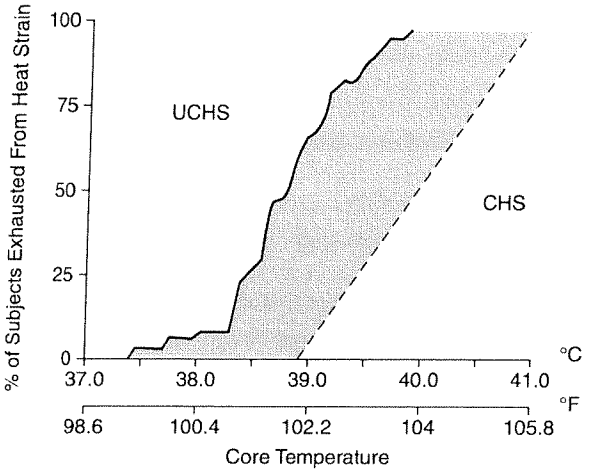

FIGURE 26.9 Relationship between core temperature and incidence of exhaustion from heat strain during exercise in compensable heat stress (CHS) and uncompensable heat stress (UCHS). *(Adapted from Sawka MN, Young AJ. Physical exercise in hot and cold climates. In: Garrett WE, Kirkendall DT, eds. Exercise and Sports Science. Philadelphia: Lippincott Williams & Wilkins, 2000:389.)*

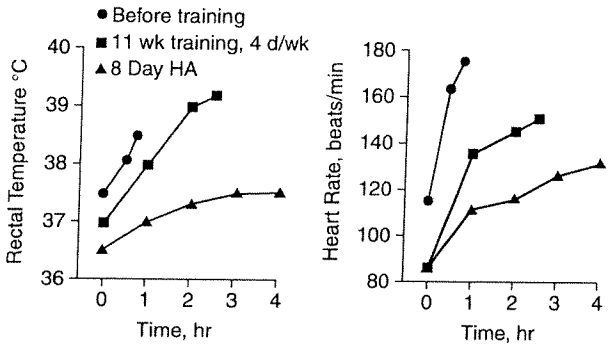

FIGURE 26.10 Core temperature and heart rate responses during standardized exercise heat stress when untrained and not acclimatized to heat, after 11 weeks of physical training and after 8 days of heat acclimatization. *(Adapted from Cohen, JS, Gisolfi CV. Effects of interval training on work-heat tolerance of young women. Med Sci Sports Exerc 1982;14:46–52.)*

nervous system component contributing to the reduced exercise performance of individuals exercising in hyperthermic conditions.

Psychological strain is probably an important factor determining an individual's desire to continue exercise in the heat. Thermal discomfort is influenced by factors including skin temperature and wetness (10), while rated perceived exertion is influenced by both of these factors as well as heart rate. It is doubtful that high core temperature alone can be sensed, but high brain temperature might induce neurochemical changes that modify perception of strain. During exercise in the heat, skin temperature, wetness, and heart rate will increase. Athletes' individual tolerance to such perceptual cues will most likely influence their exercise capabilities.

Exercise Training and Heat Acclimatization

Dynamic Exercise Training

Dynamic exercise training in a temperate climate with emphasis on improved aerobic performance can reduce physiological strain and improve exercise capabilities in the heat, but such exercise training programs alone cannot replace the benefits of heat acclimatization (45,46). Figure 26.10 depicts an exercise training program with heat acclimatization on reducing physiological strain and improving endurance during exercise heat stress (47). After completing an initial (pretraining) exercise heat test (4 h at about 35% maximal aerobic power in hot, dry conditions) the subjects completed the training program (1 h a day, 4 times per wk for 11 wk in temperate conditions), then again completed the exercise heat test. Finally, subjects completed a heat acclimatization program (35% maximal aerobic power, 4 h per day for 8 days) then again completed the exercise heat test. The exercise training reduces physiological strain and improves endurance, but these improvements are modest compared to those obtained by heat acclimatization.

Dynamic exercise training in temperate climates can improve heat loss responses during exercise heat stress, but not to the same magnitude as is induced by heat acclimatization (45,46). Figure 26.11 illustrates the impact of exercise training and heat acclimatization on improving sweating responses. Local sweating responses during exercise heat stress were measured on untrained and not acclimatized subjects, after 10 days of exercise training (70–80% maximal aerobic power in temperate conditions), and after 10 days

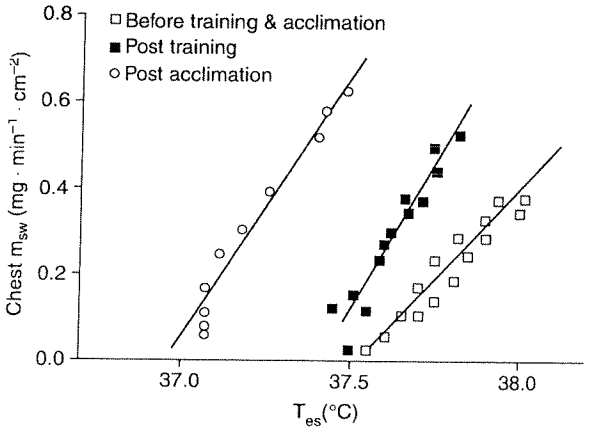

FIGURE 26.11 Local sweating responses during standardized exercise heat stress when untrained and unacclimatized to heat, after completing a physical training program, and after completing a heat acclimatization program. *(From Roberts MF, Wenger CB, Stolwijk JAJ, et al. Skin blood flow and sweating changes following exercise training and heat acclimation. J Appl Physiol 1977;43:135.)*

of heat acclimatization (1 hour at 50% maximal aerobic power in hot, wet conditions) (48). Exercise training increased the slope of the sweating response, but heat acclimatization markedly reduced threshold temperature to initiate sweating as soon as core temperature started to rise. The net effect of heat acclimatization is a substantially increased sweating rate for a given core temperature during exercise. The skin blood flow responses change in a similar manner to sweating after exercise training and heat acclimatization. Clearly, exercise training can improve heat loss responses; however, these improvements are modest compared to those obtained by heat acclimatization.

For exercise training to improve thermoregulation and exercise capabilities in the heat, the training sessions must produce a substantial elevation of core temperature and sweating rate (45). To achieve optimal improvement in thermoregulation with aerobic exercise training in temperate climates, the intensity should be at least 50% of maximal aerobic power with a minimal duration of 1 wk and for perhaps as long as 8 wk (45).

Heat Acclimatization

Heat acclimatization induces biological adjustments that reduce the negative effects of heat stress. Heat acclimatization develops through repeated heat exposures that are sufficiently stressful to elevate both core and skin temperatures and provoke perfuse sweating. The biological adjustments are mediated by integrated changes in thermoregulatory control, fluid balance, and cardiovascular responses. In addition to these systemic adaptations, emerging research information demonstrates that cellular adaptations occur (49).

The magnitude of biological adaptations induced by heat acclimatization depends largely on the intensity, duration, frequency, and number of heat exposures. Exercise in the heat is the most effective method for developing heat acclimatization; however, even resting in the heat results in limited acclimatization. Usually, about 7–10 days of heat exposure is needed to induce heat acclimatization. Optimal heat acclimatization requires a minimum daily heat exposure of about 2 h (can be broken into two 1-h exposures) combined with dynamic exercise that requires cardiovascular endurance rather than resistance training for development of muscular strength. Individuals in training should gradually increase the exercise intensity or duration each day of heat acclimatization.

During the initial exercise heat exposure, physiological strain is high, as manifested by elevated core temperature and heart rate. The physiological strain induced by the same exercise heat stress decreases each day of acclimatization. Most of the improvements in heart rate, skin and core temperatures, and sweat rate are achieved through daily exercise in a hot climate during the first week of exposure. The heart rate reduction develops most rapidly in 4–5 days. After 7 days, the reduction in heart rate is virtually complete. The thermoregulatory benefits from heat acclimatization are generally thought to be complete after 10–14 days of exposure (46). However, improvements in physiological tolerance may take longer (39).

Heat acclimatization is transient and gradually disappears if not maintained by continued repeated heat exposure (45,46). The benefits of heat acclimatization will be retained for about 1 week and then decay, with about 75% lost by about 3 wk once heat exposure ends. A day or two of intervening cool weather will not interfere with acclimatization to hot weather. The heart rate improvement, which develops more rapidly during acclimatization, is also lost more rapidly than thermoregulatory responses.

Table 26.4 provides a brief description of the actions of heat acclimatization. Heat acclimatization improves thermal comfort and submaximal aerobic exercise capabilities. These benefits of heat acclimatization are achieved by improved sweating and skin blood flow responses, better fluid balance and cardiovascular stability, and a lowered metabolic rate (2). Heat acclimatization is specific to the climate (desert or jungle) and physical activity level. However, heat acclimatization to a desert or jungle climate can markedly improve exercise capabilities in the other hot climate.

TABLE 26.4 Actions of Heat Acclimatization

Thermal Comfort Improved	Exercise Performance Improved
Core temperature reduced	*Cardiovascular stability improved*
Sweating improved	Heart rate lowered
Earlier onset	**Stroke volume increased**
Higher rate	Blood pressure better defended
Redistribution (*jungle*)	Myocardial compliance improved
Hidromeiosis resistance (*jungle*)	Fluid balance improved
	Thirst improved
Skin blood flow improved	Electrolyte loss (sweat, urine) reduced
Earlier onset	Total body water increased
Higher rate (jungle)	**Plasma (blood) volume increased,** better defended
Metabolic rate lowered	Acquired thermal tolerance improved

Heat acclimatization does not improve maximal exercise performance. For example, heat stress–mediated reductions in maximal aerobic power are not abated by heat acclimatization (35). In addition, heat acclimatization probably does not alter the maximal core temperature a person can tolerate during exercise during CHS (50,51). There is some evidence, however, that persons who live and physically train over many weeks in the heat may be able to tolerate higher maximal core temperatures during UCHS (39).

The effect of heat acclimatization on submaximal exercise performance can be quite dramatic, such that acclimatized subjects can easily complete tasks in the heat that earlier were difficult or impossible (Fig. 26.10). Heat acclimatization mediates improved submaximal exercise performance by reducing body temperature and physiological strain (Fig. 26.10). The three classical signs of heat acclimatization are lower heart rate and core temperature and higher sweat rate during exercise heat stress. Skin temperatures are often lower after heat acclimatization, and thus dry heat loss is less (or if the environment is warmer than the skin, dry heat gain is greater). To compensate for the changes in dry heat exchange, an increase in evaporative heat loss is necessary to achieve heat balance.

After acclimatization, sweating starts earlier and at a lower core temperature; that is, the core temperature threshold for sweating is decreased (Fig. 26.11). The sweat glands also become resistant to hidromeiosis and fatigue, so that higher sweat rates can be sustained. Sweating rate is often increased by the second day of heat acclimatization. Earlier and greater sweating improves evaporative cooling (if the climate allows evaporation) and reduces body heat storage and skin temperature. Lower skin temperatures decrease the cutaneous blood flow required for heat balance (because of greater core-to-skin temperature gradient) and reduce cutaneous venous compliance, so that blood volume is redistributed from the peripheral to the central circulation. All of these factors reduce cardiovascular strain and enhance exercise heat performance.

The effects of heat acclimatization on cardiac output responses to exercise heat stress are not clearcut. Some studies report little change in cardiac output, while most find an increase in cardiac output during submaximal exercise performed after heat acclimatization as compared to before it. Heart rate is much higher and stroke volume is lower (than in temperate conditions) during exercise heat stress on the first day of heat acclimatization. Thereafter, heart rate begins to decrease as early as the second day. These changes are rapid at first but continue more slowly for about a week. Probably numerous mechanisms participate, and their relative contributions will vary, both over the course of the program and also among subjects (2). These mechanisms include (*a*) improved skin cooling and redistribution of blood volume, (*b*) plasma volume expansion, (*c*) increased venous tone from cutaneous and noncutaneous beds, and (*d*) reduced core temperature. In addition, myocardial changes from heat acclimatization include increased compliance and isoenzyme transition, reducing the myocardial energy cost (52).

Fluid balance improvements from heat acclimatization include better matching of thirst to body water needs, increased total body water, and increased blood volume (53,54). Most but not all studies report that heat acclimatization increases total body water (54). The magnitude of increase often approximates 2.0 L, or about 5% of total body water. This increase is well within the measurement resolution for total body water and thus appears to be a real physiological phenomenon. The division of the total body water increase between intracellular fluid and extracellular fluid varies: studies have reported that extracellular fluid accounts for greater, equal, and smaller amounts of the percentage increase compared to intracellular fluid after heat acclimatization (54). Measures of extracellular fluid have relatively high variability, and therefore trends for such small changes are difficult to interpret. The extent to which intracellular fluid increases is unclear, because typically it is calculated as the difference between total body water and extracellular fluid, and thus measurement variability inherent in both these techniques is compounded in the calculation of intracellular fluid. If total body water and extracellular fluid increase after heat acclimatization, then expansion of plasma volume might be expected.

Plasma volume expansion is usually but not always present after repeated heat exposure and heat acclimatization (55). Erythrocyte volume does not appear to be altered by heat acclimatization. Heat acclimatization studies report that plasma volume expansion generally ranges from about 5% to about 30%, and the magnitude of increase somewhat depends on (*a*) whether the person is at rest or performing exercise, (*b*) the day of heat acclimatization, and (*c*) the hydration state when measurements are made. Plasma volume expansion seems to be greatest during upright exercise on about the fifth day of heat acclimatization in fully hydrated persons who are living in the heat.

The mechanisms responsible for this hypervolemia are unclear, but they include an increase in extracellular fluid mediated by retention of crystalloids (primarily sodium chloride) and perhaps an increase in plasma volume selectively mediated by the oncotic effect of increased volume of intravascular protein (no change in content) (54). The increase in total body water can be explained in part by increased aldosterone secretion and/or renal sensitivity to a given plasma concentration. An unacclimatized person may secrete sweat with a sodium concentration of 60 $mEq \cdot L^{-1}$ or higher and if sweating profusely, can lose large amounts of sodium. With acclimatization, the sweat glands conserve sodium by secreting sweat with a sodium concentration as low as 10 $mEq \cdot L^{-1}$. This salt-conserving effect of acclimatization depends on the hormone aldosterone, which is secreted in response to exercise and heat exposure as well as to sodium depletion. The conservation of salt also helps to maintain the number of osmoles in the extracellular fluid and thus to maintain or increase extracellular fluid volume.

Acquired Thermal Tolerance and Molecular Changes

Individuals who are repeatedly exposed to the hyperthermia of exercise can become more resistant to exertional heat injury and stroke. The repeated heating of body tissues results in acquired thermal tolerance, cellular changes resulting from severe nonlethal heat exposure that allow the organism to survive a subsequent otherwise lethal heat exposure (1,56). Acquired thermal tolerance and heat acclimatization are complementary, as acclimatization reduces heat strain and thermal tolerance increases survivability in a given heat load. For example, rodents with fully developed thermal tolerance can survive 60% more heat load than what would have been initially lethal (57).

Acquired thermal tolerance is associated with heat shock proteins (HSP) binding to denatured or nascent cellular polypeptides and providing protection and accelerating repair from heat stress, ischemia, monocyte toxicity, and ultraviolet radiation in cultured cells and animals. These HSP are grouped into families by molecular mass (56). Each has a variety of cellular locations and functions that include processing of stress-denatured proteins, management of protein fragments, maintenance of structural proteins, and chaperone of proteins across cell membranes. For example, HSP 27 (sometimes referred to as sHSP), resides in the cytosol and nucleus and has antiapoptotic and microfilament stabilization functions. The HSP 70 family (HSP 72, 73, 75, 78) resides in the cytosol and nucleus (HSP 72 and 73), endoplasmic reticulum (HSP 78), and mitochondria (HSP 75) and has molecular chaperone (HSP 73,75, 78), cytoprotection (HSP 72) and antiapoptotic (HSP 73) functions.

The HSP responses increase within several hours of the stress and last for several days after the exposure. After the initial heat exposure, mRNA levels peak within an hour, and subsequent HSP synthesis depends upon both severity of heat stress and cumulative heat stress (57). Both heat exposure and high-intensity dynamic exercise elicit HSP synthesis; however, the combination of dynamic exercise and heat exposure elicits a greater HSP response than either stressor independently (58). In addition, HSP responses vary with specific tissue, as brain and liver demonstrate greater responses than skeletal muscle. The cellular mechanisms mediating thermal tolerance are not fully understood, but it seems probable that the stress kinase pathways (59) and other cellular systems contribute (60,61).

Fluid and Electrolyte Imbalances

Water balance represents the net difference between water intake and loss. During exercise in the heat, water losses primarily occur from respiration and sweating. Renal water losses will be minimal and respiratory water losses will usually be approximately offset by metabolic water production (62). Therefore, sweat losses determine most of a person's water needs. Also, since sweat contains electrolytes (primarily sodium and chloride), prolonged periods of high sweat losses can lead to electrolyte deficits.

A person's sweating rate depends on the climatic conditions, clothing, and exercise intensity. Persons in desert and tropical climates often have sweating rates of 0.3–1.0 L · h^{-1} while performing occupational activities. Competitive marathon runners' sweating rates average about 0.8 L · h^{-1} (range 0.7–1.4 L · h^{-1}) and about 1.2 L · h^{-1} (range 0.9–2.8 L · h^{-1}) in cool to temperate and warm to hot weather, respectively (63). Active individuals, however, have high sweating rates only for several hours per day, and the remainder of time, when more sedentary, they have lower sweating rates. For competitive athletes training in temperate and hot weather, daily fluid requirements may range from 3–5 L and 4–10 L, respectively (62).

Dehydration

Persons dehydrate (sustain a body water deficit) during exercise because of unavailability of fluid or a mismatch between thirst and body water requirements. In these instances, the person starts the exercise task with normal total body water and dehydrates over time. This scenario is common for most athletic and occupational settings; however, sometimes the person starts exercise with a body water deficit. For example, in several sports (e.g., boxing, power lifting, wrestling) athletes purposefully dehydrate to compete in lower weight classes. Also, persons medicated with diuretics may be dehydrated prior to exercise. Dehydration from water deficit without excessive sodium chloride loss is the most commonly seen type during relatively short-term exercise in the heat if a normal diet is consumed. If the person has a large sodium chloride deficit, the extracellular fluid volume will contract and cause salt depletion dehydration. If the sodium chloride deficit is combined with excessive water consumption, hyponatremia, or water intoxication, can more easily occur (64).

Regardless of the approach used to induce dehydration, there is great similarity in altered physiological function and performance consequences. Dehydration can decrease dynamic exercise performance, including maximal aerobic power (54). Dehydration by more than 2% of body weight degrades endurance exercise, especially in heat (65). The magnitude of the performance decrement is variable and probably depends on the individual, the environment, and the exercise mode. However, for a given person and event, the greater the dehydration level (after achieving the threshold for performance degradation), the greater the performance decrement. Dehydration probably does not alter muscle strength but sometimes has been reported to reduce anaerobic performance (66). In addition, dehydration of more than 2% often adversely influences cognitive function in the heat; however, this area requires more research (62).

Physiological factors that contribute to dehydration-mediated performance decrements include increased hy-

perthermia, increased cardiovascular strain, altered metabolic function, and perhaps altered central nervous system function. Though each factor is unique, evidence suggests that they interact to contribute in concert, rather than in isolation, to degrading exercise performance. The relative contribution of each factor may differ depending on the endurance event, environmental conditions, and athlete prowess, but elevated hyperthermia probably acts to accentuate the performance decrement (65).

Dehydration increases core temperature responses during exercise in temperate and hot climates (54). A body water deficit of as little as 2% of body weight can elevate core temperature during exercise. As the magnitude of water deficit increases, there is a concomitant graded elevation of core temperature during exercise in the heat. The magnitude of core temperature elevation approximates 0.2°C for every percent body weight lost, and it is influenced by the environment. The hotter the environment and greater the evaporative heat loss requirement, probably the greater the core temperature elevation. Dehydration not only elevates core temperature; it negates the core temperature advantages conferred by high aerobic fitness and heat acclimatization. In addition, dehydration lowers the core temperature that can be tolerated before exhaustion from heat strain during uncompensable heat stress.

When a person is dehydrated, the elevated core temperature responses during exercise result from reduced heat dissipation. Both sweating and skin blood flow are reduced for a given core temperature during dehydration (54). For both thermoregulatory effector responses (sweat secretion and active cutaneous vasodilation), threshold temperature (body temperature at initiation of heat loss response) is increased and sensitivity (increase in heat loss for a given increase in body temperature) is reduced during dehydration. For thermoregulatory sweating, the increased threshold temperature and reduced sensitivity are proportional to the level of dehydration. Therefore, the degraded sweating response represents an attempt to conserve body water losses. Whole-body sweating is usually either reduced or unchanged during exercise at a given metabolic rate in the heat. However, even when dehydration is associated with no change in whole-body sweating rate, core temperature is usually elevated, so that whole-body sweating rate for a given core temperature is lower when dehydrated.

Both the singular and combined effects of plasma hyperosmolality and hypovolemia mediate the reduced heat loss response during exercise heat stress (67). Plasma hyperosmolality with no change in blood volume can increase core temperature by reducing heat loss during rest or exercise in the heat. In addition, reestablishment of blood volume that is still hyperosmotic does not alter the core temperature elevation compared to responses to dehydration during prolonged exercise in the heat. Plasma volume reductions with no change in osmolality can increase core temperature and impair heat loss during exercise in the heat (67).

Dehydration increases cardiovascular strain during dynamic exercise in temperate and hot climates (54). During submaximal dynamic exercise with little heat strain, dehydration elicits an increase in heart rate and decrease in stroke volume but usually no change in cardiac output relative to euhydration levels. Heat stress and dehydration, however, have additive effects on cardiovascular strain. If evaporative cooling is reduced, the skin temperature will be elevated, which increases compliance (superficial cutaneous veins) and acts to transfer blood from central to peripheral circulation. This transfer of blood volume, combined with the hypovolemia, acts to reduce cardiac filling and stroke volume, making it more difficult to sustain sufficient cardiac output to support muscle metabolism. The inability to sustain sufficient cardiac output provides a cardiovascular limitation to exercise in the heat. During submaximal dynamic exercise with moderate or severe heat strain, dehydration (3–4% body weight) leads to a decrease in cardiac output. The dehydration-mediated cardiac output reduction (below euhydration levels) during heat stress is greater during high-intensity (65% maximal aerobic power) than low-intensity (25% maximal aerobic power) exercise. In addition, severe water deficit (7% body weight) in the absence of heat strain can reduce cardiac output during submaximal exercise.

During severe exercise heat stress when dehydration mediates a reduction in cardiac output, the skeletal muscle blood flow can also be decreased (30). Despite having decreased muscle blood flow, substrate delivery and lactate removal are not impaired by dehydration. However, dehydration can reduce free fatty acid uptake and increase muscle glycogen utilization and muscle lactate production during intense exercise. The dehydration-associated increase in muscle glycogen utilization during exercise is probably mediated by elevated catecholamine levels.

Dehydration might reduce exercise performance through mechanisms mediated by the central nervous system. As discussed earlier, dehydration elevates body temperature, and there is evidence that hyperthermia might diminish the drive to exercise and reduce exercise tolerance time.

Hypervolemia and Hyperhydration

Hypervolemia, or blood volume expansion, can improve thermoregulation and exercise performance in the heat if the erythrocyte volume alone or erythrocyte volume and plasma volume together are expanded (54,68). Plasma volume expansion alone does not provide a thermoregulatory benefit, but it does reduce cardiovascular strain (54). Studies that only expanded erythrocyte volume have demonstrated small thermoregulatory benefits, but when both erythrocyte volume and plasma volume were expanded, more substantial benefits were observed. The former experiments were on unacclimatized subjects, and the latter experiments were on heat-acclimatized subjects, so it is unclear whether accentuated thermoregulatory benefits resulted from simultaneous

expansion of both vascular volumes or the subjects' acclimatization state (68).

Hyperhydration, or above-normal body water, has been suggested to improve, above euhydration levels, thermoregulation and exercise heat performance (54). The concept that hyperhydration might be beneficial for exercise performance arose from the recognition of the adverse consequences of dehydration. It was theorized that an increase in body water might reduce cardiovascular and thermal strain of exercise by expanding blood volume and reducing blood tonicity, thereby improving exercise performance. In some studies that have evaluated hyperhydration effects on thermoregulation in the heat, smaller core temperature elevations during exercise were observed with hyperhydration, but those studies generally have confounding factors in experimental procedures. Studies that were carefully controlled for these confounding factors have not observed any thermoregulatory advantages with either water hyperhydration or glycerol hyperhydration during exercise heat stress (54).

Hyponatremia

Symptomatic hyponatremia (serum sodium concentration of 125–130 mEq · L^{-1}) has been observed during prolonged marathon and ultramarathon competition, military training, and recreational activities (64). Symptomatic hyponatremia is very rare, and hospitalizations occur at a rate of less than 1 per 100,000 person years for U.S. Army soldiers (69). The severity of symptoms is probably related to the serum sodium concentration and the rapidity with which it develops. If hyponatremia develops over many hours, there may be less brain swelling and less adverse symptoms than when changes are rapid. Hyponatremia usually develops because a person drinks excessively large quantities of hypotonic fluids for many hours. However, other factors, such as excessive sodium losses, nausea (which increases vasopressin levels), and heat or exercise stress (which reduce renal blood flow and urine output), can contribute when excessively large volumes of fluids are consumed. For a person to become hyponatremic, total body water usually must be markedly increased, and excessive intake of hypotonic fluids and/or significant sodium loss must occur (64).

Table 26.5 presents the predicted serum sodium response to prolonged exercise (90-km ultramarathon foot race) for two individuals of low and average body mass, with three sweat sodium concentrations, who replace their sweat losses with sodium-free fluid (64). For the example, it is assumed that total body water is 63% of body mass and that water distributes within the extracellular fluid and intracellular fluid until osmotic equilibrium is reached. This analysis illustrates that sweat sodium losses are an important contributor to the reduction of serum sodium when water or a sodium-free solution is used to replace sweat lost during exercise. In the example provided, if a 70-kg man had sweat sodium concentrations of 25, 50, and 75 mEq · L^{-1} and drank sufficient sodium-free water to replace all of the 8.6 L of sweat loss, serum sodium would be expected to decline about 5, 10, and 15 mEq · L^{-1}. However, to lower the serum sodium to the average value reported for individuals with symptomatic hyponatremia (121 mEq · L^{-1}), the 70-kg man would still have to accrue a fluid excess of 5.1, 3.3, and 1.6 L at the low, moderate, and high sweat sodium concentrations, respectively. Example calculation for a 70-kg person with low sweat sodium: x / 44 L TBW × 135 mEq · L^{-1} / 121 mEq · L^{-1} = 49.1 L; then 49.1–44 = 5.1 L.

This analysis also illustrates that smaller individuals with similar sweat sodium concentrations will develop symptoms in the presence of less fluid excess to dilute serum sodium than larger persons. Therefore, if a group of people are drinking at the same rate (as may happen if they follow drinking schedules that are not adjusted for body mass), the individuals with smaller body mass will dilute their extra-

TABLE 26.5 Predicted Serum Sodium Values to Prolonged Exercise[a]

Body mass, kg	50			70		
Total body water, L	31.5			44		
extracellular fluid, L	12.5			17.5		
Serum [Na^+], mEq · L^{-1}	140			140		
extracellular fluid Na^+, mEq	1750			2450		
Running velocity, km · h^{-1}	10			10		
Sweat loss, liters	6.1			8.6		
Calculations of serum sodium dilution if water intake is equal to sweat loss						
Sweat [Na^+], mEq · L^{-1}	25	50	75	25	50	75
Na^+ loss, mEq	153	305	458	215	430	645
Δ TBW osmol · L^{-1}	4.9	9.7	14.5	4.9	9.8	14.7
Serum [Na^+], mEq · L^{-1}	135.1	130.3	125.5	135.1	130.2	125.3
Fluid excess to dilute serum Na^+ to 121 mEq · L^{-1}	3.7	2.4	1.2	5.1	3.3	1.6

[a]90-km ultramarathon foot race for two body masses, when body mass is preserved by ingestion of sodium-free water.

cellular sodium levels more quickly and further than those with larger body mass. On the other hand, to produce the magnitude of sodium dilution observed in symptomatic hyponatremia cases, persons who are larger than 70 kg either would have to substantially overdrink relative to sweating rate or would have to have an abnormally high sweat sodium concentration.

Environmental Heat Stress

Sports and occupational medicine communities commonly use WBGT to quantify environmental heat stress and set limits on exercise in planned strategies designed to minimize the risk of serious exertional heat illness. WBGT is an empirical index of climatic heat stress: outdoor WBGT = 0.7 natural wet bulb + 0.2 black globe + 0.1 dry bulb. Indoor WBGT = 0.7 natural wet bulb + 0.3 black globe. (Natural wet bulb is a stationary wet-bulb thermometer exposed to the sun and prevailing wind. The black globe thermometer consists of a 6-inch hollow copper sphere painted flat black on the outside, containing a thermometer with its bulb at the center of the sphere.) High WBGT values can be achieved either through high air (dry bulb) temperature and solar load, as reflected in black-globe temperature, or through high humidity, as reflected in high wet-bulb temperature. However, WBGT underestimates heat stress risk for humid conditions, so different guidance tables should be used in low-, moderate-, and high-humidity climates. Table 26.6 provides the relative risk of excessive hyperthermia and possibly serious exertional heat illness for an individual exercising in the heat or an athlete competing in running events while lightly clothed and sustaining a high metabolic rate (70).

Exertional Heat Illness

Serious exertional heat illnesses occupy a continuum of increasing severity of heat exhaustion, heat injury, and heat stroke (70). Heat exhaustion is defined as a mild to moderate illness characterized by inability to sustain cardiac output with moderate (>38.5°C) to high (>40°C) body temperature resulting from strenuous exercise and environmental heat exposure. Exertional heat injury is defined as a moderate to severe illness characterized by organ (e.g., liver, renal) and tissue (e.g., gut, muscle) injury with high body temperature resulting from strenuous exercise and environmental heat exposure. Exertional heat stroke is defined as a severe illness characterized by central nervous system dysfunction, organ (e.g., liver, renal), and tissue (e.g., gut, muscle) injury with high body temperatures resulting from strenuous exercise and environmental heat exposure.

Risk factors for serious exertional heat illness include lack of heat acclimatization, low physical fitness, dehydration and high body fat or mass, and certain medications (Table 26.7). However, serious exertional heat illness can occur in low-risk persons who are practicing sound heat mitigation procedures. Exertional heat injury and stroke often occur under conditions the victim has been exposed to many times before or while others are concurrently being exposed to the same condition without incident. This suggests that these victims were inherently more vulnerable that day and/or some unique event triggered the heat injury.

Many victims of exertional heat stroke were sick the previous day. Exertional heat stroke often occurs during the initial hours of exercise heat stress and may not occur during the hottest part of the day. These facts suggest that on that day, the victim began the exercise already heat stress compromised. Fever and inflammatory responses from muscle injury may adversely influence thermoregulation and may help mediate subsequent exertional heat injury or stroke. Gastrointestinal problems will induce dehydration and increase the risk of serious exertional heat illness or may indicate previous heat injury (e.g., gut ischemia causing endotoxin leakage). Evidence suggests that some cases of exertional heat injury or stroke might be explained by an association between susceptibility to malignant hyperthermia and exertional heat stroke.

Recent research shows that exertional heat injury (unlike heat shock *in vitro*), produces a broad spectrum of

TABLE 26.6 Risk of Hyperthermia and Possible Exertional Heat Illness for a Typical Marathon Racer[a]

Risk of Hyperthermia	Color Code (flag)	WBGT, °C (°F) RH, 100%	WBGT, °C (°F) RH, 75%	WBGT, °C (°F) RH, 50%
Excessive	Black	>28	>29	>33
High	Red	24–28 (73–82)	26–29 (77–85)	28–33 (82–92)
Moderate	Amber	18–23 (65–72)	20–25 (68–76)	24–27 (75–81)
Low	Green	<18 (<65)	<20 (<68)	<24 (<75)
Low (some risk of hypothermia)	White	<10 (<50)	<10 (<50)	<10 (<50)

[a]Based on wet-bulb globe temperature (WBGT) and relative humidity (RH).

TABLE 26.7 Drugs Implicated in Intolerance to Heat Stress

Drug or Drug Class	Proposed Mechanism of Action
Anticholinergics (Atropine)	Impaired sweating
Antihistamines	Impaired sweating
Gluthemide (Doriden)	Impaired sweating
Phenothiazines (antipsychotic drugs, e.g., chlorpromazine [Thorazine], trifluoperazine [Stelazine], perphenazine [Trilafon])	Impaired sweating, (possibly) disturbed hypothalamic temperature regulation
Tricyclic antidepressants, e.g., imipramine, amitriptyline	Impaired sweating, increased motor activity and heat production
Amphetamines, cocaine	Increased psychomotor activity, activated vascular endothelium
Ergogenic stimulants, e.g., ephedrine, ephedra	Increased heat production
Lithium	Nephrogenic diabetes insipidus and water loss
Diuretics	Salt depletion and dehydration
β-Blockers, e.g., propranolol, atenolol	Reduced skin blood flow, reduced blood pressure
Ethanol	Diuresis, possible effects on intestinal permeability

gene expression changes, including interferon-induced sequences (61). Elevated levels of circulating interferon-γ are reported in victims of classic heat stroke, and it can increase cellular mortality by apoptosis after heat shock. A prior incident of cellular heat shock or some other unknown event might mediate expression of interferon genes and subsequently elevated circulating interferon-γ levels. Expression of this or another cytokine might contribute to the pathogenesis of exertional heat injury (71).

Figure 26.12 provides a diagram of the possible progression of heat strain to exertional heat injury and/or stroke (71). The hyperthermia and cardiovascular responses to exercise heat stress can result in reduced perfusion of the intestine and other body tissues, resulting in ischemia and excessively high tissue temperatures (heat shock, >41°C). The magnitude and duration of the heat shock will influence whether the cell responds by adaptation (acquired thermal tolerance), injury, or death (apoptotic or necrotic) (56,59). This can result in a variety of systemic coagulation and inflammatory responses (71). It is suspected that the inflammatory response is primed (e.g., leukocytosis, expression of inflammatory cytokines) so that a subsequent exposure to severe exercise heat induces an accentuated acute-phase response. This exaggerated acute-phase response could mediate fever (in addition to exercise hyperthermia) and/or enhance likelihood of tissue injury and cellular death.

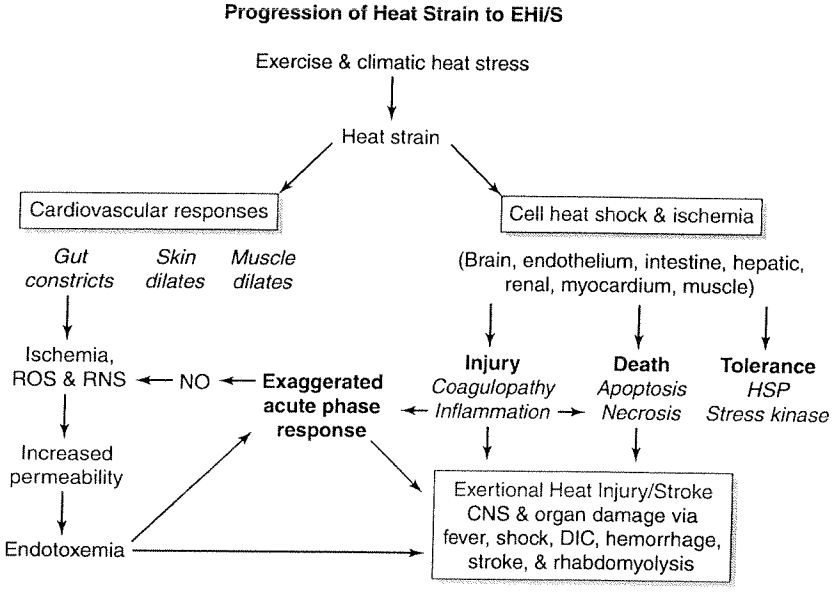

FIGURE 26.12 Possible pathogenesis for progression of exercise heat strain to exertional heat injury and stroke. *(Adapted from Bouchama A, Knochel P. Heat stroke. N Engl J Med 2002;346:1982.)*

The severity of heat injury is often not apparent on presentation. Individuals performing or competing in strenuous activities in hot weather who exhibit signs and symptoms of serious exertional heat illness (e.g., unsteady gait; sweaty, flushed skin; dizziness; headache; tachycardia; paresthesias; weakness; nausea; cramps) should be immediately evaluated for mental status, core (rectal) temperature, and vital signs. Poor or worsening mental status (ataxia, confusion) is a true medical emergency, and these individuals need rapid intervention and evacuation to a medical treatment facility.

Regardless of the pathogenesis, serious exertional heat illness poses a risk to healthy persons who are active in warm weather. Guidelines for the prevention and treatment of serious exertional heat illness can be readily obtained (http://usariem.army.mil/HeatInjury.htm and http://www.naspem.org/pos_stmts/) and should be followed.

All persons suspected of having exertional heat injury or stroke must have early initiation of cooling in the field. Delay in cooling probably is the single most important factor leading to death or residual serious disability. The patient should lie down, and as much clothing should be removed as is practical. Body cooling should be initiated and continued until core temperature is less than 38.5°C by the most practical means as quickly as possible. Immersion in cool or iced water with skin massage is the most rapid method, but ice sheets and ice packs can be effective.

Cold Stress

Environmental Cold Stress

The principal determinants of cold stress during outdoor events in cold weather are air temperature and wind speed. Most body heat loss during cold exposure occurs via conductive and convective mechanisms, so when ambient temperature is colder than body temperature, the thermal gradient favors body heat loss, and wind exacerbates heat loss by facilitating convection at the body (5). Wind chill charts and tables have been constructed to depict, for every combination of air temperature and wind speed, a corresponding temperature in calm air (i.e., no wind) that is calculated to produce the same theoretical heat flow through bare skin, and this value is the equivalent chill temperature for the temperature and wind combination (72). The effect of increasing wind speed with decreasing air temperature on equivalent chill temperature is illustrated in Figure 26.13.

Although widely reported and broadly accepted as an overall cold stress index, wind chill temperatures really only estimate the danger of cooling the exposed flesh of sedentary persons. Wind chill effects are greatly reduced by wearing windproof clothing and/or engaging in strenuous exercise. Further, wind chill provides no meaningful estimate of the risk of hypothermia. Thus, wind chill tables probably somewhat exaggerate the risk of cold injury during endurance competition, and events in which participants are properly dressed

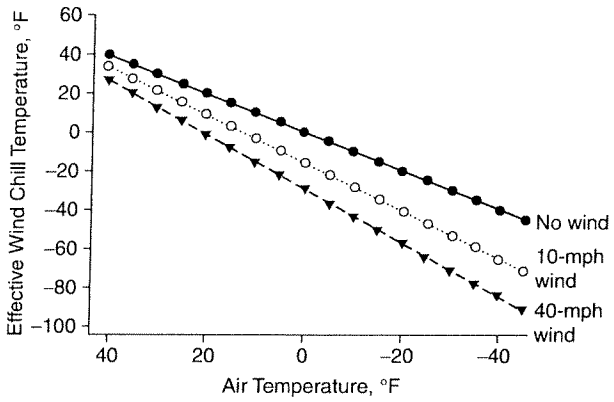

FIGURE 26.13 Effects of increasing wind speed with decreasing air temperature on equivalent chill temperature (ECT). Prepared from wind chill index values extracted from the National Weather Service Wind Chill Chart. *(Data from National Weather Service. Wind chill temperature index. Office of Climate, Water, and Weather Services, NOAA, National Weather Service, 2001 http://www.nws.noaa.gov/om/windchill/index.shtml)*

and maintain high metabolic rates need not be canceled because of the wind chill alone. Lacking a better tool, equivalent chill temperature is useful for guiding decisions regarding the need to cancel outdoor activities, but, as with the WBGT, the limitations to this approach should be appreciated. Prudence does warrant increased safety surveillance of competitors when equivalent chill temperatures fall below –30°C, since injured or fatigued athletes may be unable to sustain high metabolic rates and skin temperatures that protect them from wind chill.

As described at the beginning of this chapter, water has a much higher thermal capacity than air, and the heat transfer coefficient in water is 25 times greater than in air (3). Therefore, heat conduction away from the body is greater during exposure to a given cold air temperature when skin and clothing are wet (e.g., from rain) than when the skin is dry. With an air temperature of 5°C, heat loss in wet clothes may be double that in dry ones (73). Even so, heat loss predictions (74) indicate that core temperature of an average-sized individual performing high-intensity endurance exercise (metabolic rate of 600 W) in an air temperature of 5°C and continuous rain will not fall below 35°C for at least 7 h, and experimental observations tend to confirm those predictions (75). The enhancement of conductive heat loss is much more pronounced during full or partial immersion. During exercise in water, skin heat conductance can be 70 times greater than comparable exercise in air at the same temperature; thus, swimmers can lose considerable body heat even in relatively mild water temperatures. However, as will be explained later, anthropomorphic factors, exercise type and intensity, metabolic rate, aerobic capacity, and water temperature all interact in a complex manner to determine net thermal balance during immersion in cold water, so individuals

vary considerably with respect to the water temperature they can tolerate without a dangerous decline in core temperature during exercise.

Safe limits for allowable duration of recreation involving immersion in different water temperatures can be predicted using thermoregulatory models (76) or estimated according to actual observations of survival times following accidental immersion (77), the latter being the less conservative approach. Figure 26.14 illustrates both approaches to establishing water temperature safety limits for aquatic events. However, in both cases, expected time to death due to hypothermia or drowning due to the inability to maintain consciousness or sustain useful physical activity is used to define the limits, and this approach may be too liberal for athletic competition.

Physiological Responses to Cold

Acute Cold Exposure

Humans exhibit peripheral vasoconstriction upon exposure to cold. The resulting decrease in peripheral blood flow reduces convective heat transfer between the body's core and shell (skin, subcutaneous fat, and skeletal muscle), effectively increasing insulation by the body's shell. Since heat is lost from the exposed body surface faster than it is replaced, skin temperature declines. During whole-body exposure to cold, the vasoconstrictor response extends beyond the fingers and spreads throughout the entire body's peripheral shell. Vasoconstriction begins when skin temperature falls below about 35°C and becomes maximal when skin temperature is about 31°C or less (78). Thus, the vasoconstrictor response to cold exposure helps retard heat loss and defend core temperature but at the expense of a decline in temperature of peripheral tissue.

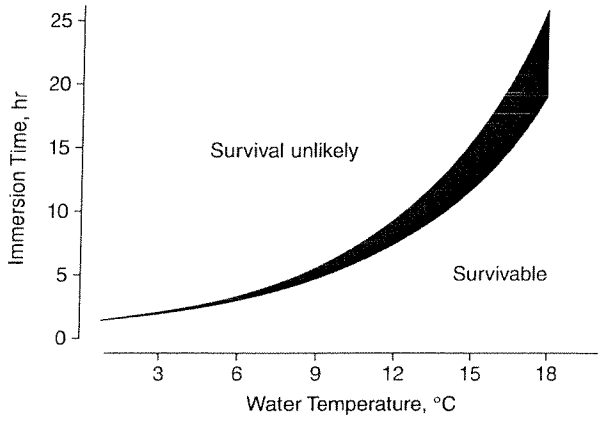

FIGURE 26.14 Approximate survival times during cold water immersion. *(From Pandolf KB, Young AJ. Assessment of environmental extremes and competitive strategies. In Shephard RJ, Astrand PO, eds. Endurance in sport. Oxford: Blackwell Science, 2000;292.)*

The vasoconstriction-induced blood flow reduction and fall in skin temperature probably contribute to the causation of peripheral cold injuries. Cold-induced vasoconstriction has pronounced effects in the hands and fingers, making them particularly susceptible to cold injury and loss of manual dexterity (79). In these areas, another vasomotor response, cold-induced vasodilation, modulates the effects of vasoconstriction. Periodic fluctuations of skin temperature follow the initial decline during cold exposure, resulting from transient increases in blood flow to the cooled finger. A similar cold-induced vasodilation in the forearm appears to reflect vasodilation of muscle as well as cutaneous vasculature (80). Originally thought to be a local effect of cooling, evidence suggests that a central nervous system mechanism medicates cold-induced vasodilation (81).

Cold exposure also elicits increased metabolic heat production in humans, which can help offset heat loss. In humans, cold-induced thermogenesis is attributable to skeletal muscle contractile activity. Humans initiate this thermogenesis by voluntarily modifying behavior, that is, increasing physical activity (e.g., exercise, increased fidgeting) or by shivering. While certain animals increase metabolic heat production by noncontracting tissue in response to cold exposure, that is, nonshivering thermogenesis, adult humans lack this mechanism.

Shivering, which consists of involuntary repeated rhythmic muscle contractions during which most of the metabolic energy expended is liberated as heat and little external work is performed, may start immediately or after several minutes of cold exposure. It usually begins in torso muscles, then spreads to the limbs (82). The intensity and extent of shivering vary according to the severity of cold stress. As shivering intensity increases and more muscles are recruited to shiver, whole-body metabolic rate increases, typically reaching about 200 to 250 W during resting exposure to cold air but often exceeding 350 W during resting immersion in cold water. Maximal shivering is difficult to quantify, but the highest metabolic rate reported in the literature to date appears to be 763 W, recorded during immersion in 12°C water, and this corresponded to 46% of that test subject's maximal aerobic power (83).

Patterns of Human Cold Acclimatization

Athletes exposed to cold weather can acclimatize, but the specifics of the physiological adjustments are modest and depend on the severity of the exposures. Cold acclimatization in persons repeatedly or chronically exposed to cold manifests in three patterns of thermoregulatory adjustments: habituation, metabolic acclimatization, and insulative acclimatization. Figure 26.15 summarizes the characteristic features of each pattern (4).

The most commonly observed acclimatization pattern is habituation. With habituation, physiological responses to cold become less pronounced than before acclimatization,

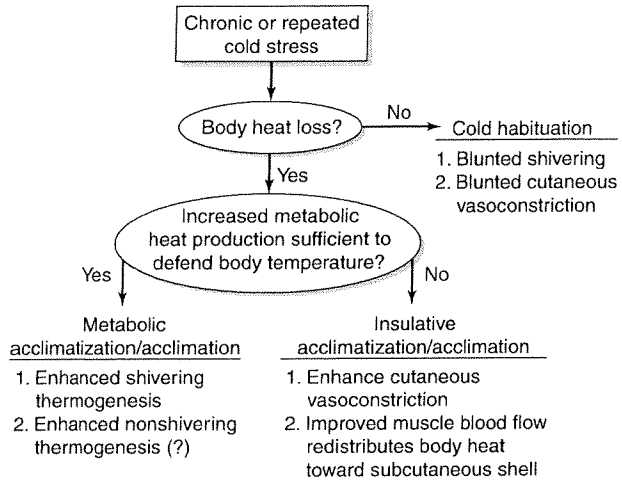

FIGURE 26.15 Theoretical flow chart to explain how humans develop different patterns of cold acclimatization. *(From Young AJ. Homeostatic responses to prolonged cold exposure: human cold acclimatization. In MJ Fregly, CM Blatteis, eds. Handbook of Physiology: Section 4: Environmental Physiology. New York: Oxford University, 1996;435.)*

and blunting of both shivering and cold-induced vasoconstriction are the hallmarks of habituation (4). Sometimes, but not always, cold-habituated persons with blunted shivering and vasoconstrictor responses to cold also exhibit a more pronounced decline in core temperature during cold exposure than nonacclimatized persons, a pattern called hypothermic habituation. Findings from different cold acclimatization studies viewed collectively (see Young [4] for a detailed review) suggest that the distinction between studies observing hypothermic habituation and those in which habituation of shivering and vasoconstriction occurred without effect on core temperature responses to cold probably reflect differences in experimental protocols rather than physiological mechanisms.

The second distinct pattern of acclimatization induced by chronic cold exposure is characterized by a more pronounced thermogenic response to cold, hence is termed metabolic acclimatization (4). Studies in which subjects appeared to demonstrate development of an enhanced thermogenic response to cold exposure seem to have observed subjects chronically (months to lifetime) exposed to relatively mild whole-body cold that was tolerated without producing hypothermia (4). Enhanced thermogenic responses to cold could arise either through an exaggerated shivering response or through development of nonshivering thermogenesis. Experimental evidence purporting to document the existence of both thermogenic adjustments has been reported (for review see Young [4]), but a critical analysis of those reports suggests little support for the development of a nonshivering thermogenic response in humans.

The third major pattern of cold acclimatization, referred to as insulative cold acclimatization, is characterized by enhanced heat conservation mechanisms (4). With insulative acclimatization, cold exposure elicits a more rapid and more pronounced decline in skin temperature and lower thermal conductance at the skin than in the unacclimatized state. The response is mediated by a pronounced vasoconstrictor response to cold, possibly due to enhanced sympathetic nervous response to cold. In addition, some data suggest that insulative cold acclimatization may also involve development of enhanced circulatory countercurrent heat exchange mechanisms to limit convective heat loss, as evidenced by the observation that before wet suits came into common usage, Korean diving women immersed in cool water exhibited less forearm heat loss than control subjects, despite the fact that concomitant forearm blood flow remained higher in the diving women. After wet suit use became widespread, Korean diving women no longer exhibited any thermoregulatory adjustments compared to control subjects, which suggests that the previous differences truly reflected adjustments to frequent exposure to cold while diving (for review see Young [4]).

The nature of the cold exposure may determine the pattern of acclimatization. In a suggested theoretical model the key determinant for cold acclimatization is the degree to which cold exposure results in body heat loss (4), and some recently reported experimental data support that concept (84). This model, illustrated in Figure 26.15, postulates that brief, intermittent cold exposures in which limited areas of the body surface are cooled and whole-body heat losses are negligible produces habituation. By contrast, repeated cold exposures prolonged and/or severe enough to preclude increased metabolic heat production from balancing body heat loss (i.e., deep body temperature declines), will induce insulative acclimatization. That model also postulates that an enhanced thermogenic capability will develop when repeated and prolonged cold exposures produce significant body heat loss, but increased body heat production can be sustained sufficiently to prevent significant core temperature declines. An alternative to that model's explanation for development of different patterns of cold acclimatization is that the metabolic, hypothermic habituative, and insulative patterns do not represent different types of cold acclimatization but are actually different stages in the development of complete cold acclimatization. Thus, initially humans respond to whole-body cold exposure by shivering, which becomes more pronounced over time; eventually, however, shivering disappears and is replaced by insulative adaptations to help limit body heat loss.

Compared to the effects of heat acclimatization, physiological adjustments to chronic cold exposure are less pronounced, slower to develop, and less practical in terms of relieving thermal strain, defending normal body temperature, and preventing thermal injury. Therefore, researchers have directed less attention to these physiological adjustments than

to effects of heat acclimatization. Nonetheless, physiologists need to appreciate these adjustments, as they may be sufficient to influence physiological responses observed during exercise and/or exposure to environmental stress, particularly under controlled experimental conditions.

Individual Factors Modifying Responses to Cold

Anthropometric Characteristics

Most variability between individuals in their thermoregulatory responses and capability to maintain normal body temperature during cold exposure is attributable to anthropometric differences. Large individuals lose more body heat in the cold than smaller individuals because they have larger body surface area, but this effect is somewhat mitigated, since a large body mass favors maintenance of a constant temperature by virtue of a greater heat content than a small body mass. In general, persons with a large ratio of surface area to mass have greater declines in body temperature during cold exposure than those with a smaller ratio (3,5).

All body tissues provide thermal resistance to heat conduction (i.e., insulation) from within the body. In resting individuals, unperfused muscle tissue provides a significant contribution to the body's total insulation. However, that contribution declines during exercise or other physical activity because increased blood flow through muscles facilitates convective heat transfer from core to the body's shell. The thermal resistivity of fat is greater than that of other tissues (3), and as illustrated in Figure 26.16, subcutaneous fat provides significant insulation against heat loss in the cold. Consequently, fat persons have smaller body temperature changes and shiver less during cold exposure than lean persons (3).

Gender-associated differences in thermoregulatory responses and ability to maintain normal thermal balance during cold exposure appear almost entirely attributable to anthropometric characteristics (3). For example, in men and women having equivalent total body mass, surface areas are similar, but the women's greater fat content enhances insulation. However, in women and men of equivalent subcutaneous fat thickness, the women have a greater surface area but smaller total body mass (and lower total body heat content) than men. Thus, while insulation is equivalent, total heat loss would be greater in the women because they have a larger surface area for convective heat flux, and body temperature would tend to fall more rapidly for any given thermal gradient unless shivering thermogenesis compensated with a more pronounced increment than in the men. This compensation may be possible when heat flux is low (mild cold conditions), but women's smaller lean body mass limits their maximal capacity for thermogenic response; therefore, a more rapid core temperature decline might occur under severely cold conditions than in men of comparable body mass (85,86).

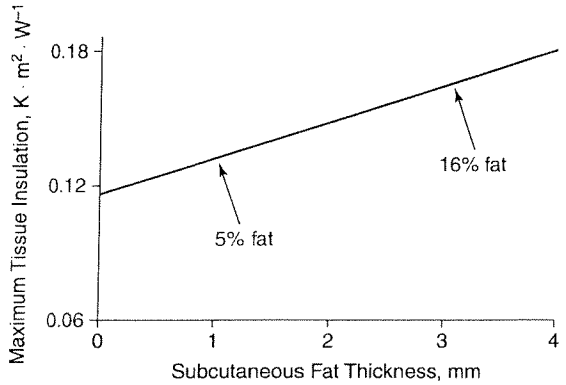

FIGURE 26.16 Tissue insulation during immersion in cold water as a function of subcutaneous fat thickness. *(Adapted from Hong SK. Pattern of cold adaptation in women divers of Korea (Ama). Fed Proc 1973;32:1618.)*

Aerobic Fitness and Training

Overall, exercise training and aerobic fitness appear to have only minor influences on thermoregulatory responses to cold. Most cross-sectional comparisons of aerobically fit and less fit persons find no relationship between maximal aerobic power and temperature regulation in cold, and in studies purportedly demonstrating a relationship, differences in thermoregulation appear more likely attributable to anthropometric differences between the aerobically fit and less fit subjects than to an effect of maximal aerobic power *per se* (87). Longitudinal studies have shown interval training to have no measurable effects on thermoregulatory response to cold (88), and while endurance training was shown to improve cutaneous vasoconstrictor response during cold water immersion, that effect had little impact on core temperature changes during cold exposure (34). The effects of resistance training programs on thermoregulatory responses to cold have not been documented, but it seems likely that any such effects would be primarily attributable to training-related changes in body composition. The primary thermoregulatory advantage provided by the increased strength and aerobic power resulting from exercise training is that the fitter individual can sustain voluntary activity at higher intensity and thus higher rates of metabolic heat production for longer periods than less fit persons during cold exposure.

Clothing

A wide variety of athletic clothing is available to provide protection during exercise and recreational activity in cold conditions. Detailed consideration of the biophysics and heat transfer properties of cold-weather athletic clothing is considered elsewhere (89) and is beyond the scope of this text. However, it should be intuitively obvious that the amount of clothing insulation required to maintain comfort and insulate against excessive body heat loss during cold-weather activity

will depend on combined effects of two opposing factors: thermal gradient for heat loss (i.e., ambient temperature) and rate of metabolic heat production (i.e., exercise intensity). Increasing clothing insulation will be required as ambient temperature decreases, while decreasing clothing insulation will be required as metabolic rate increases. Heat production during high-intensity exercise can be sufficient to prevent a fall in deep body temperature without the need for heavy clothing even when air temperature is extremely low, but athletes dressed optimally during events may be inadequately protected from cold before starting or when exercise ceases because of fatigue, injury, or completion of the event. Thus, modern cold-weather athletic clothing incorporates design features enabling adjustable insulation, such as ventilating openings and clothing layering. Further, since much of the insulation provided by cold-weather clothing is achieved by trapping stagnant air layers within the clothing or against the skin, the clothing must also provide a barrier against moisture and wind, which degrade insulation by disrupting trapped air layers. Also, even during activity of sufficiently high intensity that metabolic heat production completely obviates heavy cold-weather clothing to protect against hypothermia, exposed skin of the fingers, nose, and ears may be susceptible to freezing injuries, and clothing to protect those regions from surface cooling should be worn when wind chill conditions are extreme. Finally, since the insulation provided by cold-weather clothing adds to insulation provided by body fat and other tissues, clothing requirements will vary among individuals, depending on anthropometric factors discussed earlier.

Exercise in the Cold

Oxygen Uptake and Systemic Oxygen Transport

As the noted environmental physiologist Dave Bass once observed (90), "man in the cold is not necessarily a cold man." Thus, whether physiological responses to exercise are altered by cold environment depends on whether the interactive effects of environment, clothing, anthropometric factors, and exercise intensity are such that cold stress elicits additional physiological strain beyond that associated with the same exercise under temperate conditions.

If cold stress is severe enough to cause a significant decline in core or muscle temperature, maximal aerobic power can be reduced, compared to that measured at normal core and muscle temperatures (91). The mechanism for this effect is not definitively demonstrated, but it probably reflects an effect of low tissue temperature on contractile function of heart (91) and/or skeletal (92) muscle. An impairment of myocardial contractility could limit maximal cardiac output, thus accounting for the reduced maximal aerobic power. Further, impairment of skeletal muscle contractile function associated with muscle cooling could reduce maximal aerobic power by simply limiting demand for oxygen transport. However, there must be a threshold temperature for this effect, as exposure to cold conditions that lower core temperature 0.5°C or less have no significant effect on maximal aerobic power and associated responses (93).

Whether cold exposure influences oxygen uptake during steady-state submaximal exercise depends on whether exercise thermogenesis is sufficient to balance the rate of body heat loss to the ambient environment and maintain core and skin temperatures warm enough that shivering does not develop. During exercise at intensities too low for metabolic heat production to balance heat loss to the cold environment and prevent shivering, oxygen uptake will be higher than in warm conditions, with the increased oxygen uptake representing the added oxygen requirement for metabolism in the shivering muscles. With increasing exercise intensity, metabolic heat production rises and core and skin temperatures are maintained warmer. Therefore, the afferent stimulus for shivering declines along with the shivering-associated component of total oxygen uptake during exercise until eventually exercise metabolism becomes high enough to prevent shivering, and the steady-state oxygen uptake at that intensity and higher is the same in cold and warm conditions.

Figure 26.17 illustrates conceptually the influence of exercise intensity on steady-state submaximal oxygen uptake during exercise (94). The precise effect of exercise intensity on the shivering component of total oxygen uptake during exercise and the specific intensity at which metabolic heat production is sufficient to prevent shivering will depend on the severity of cold stress, clothing insulation, and anthropometric factors influencing body heat flux.

Cold exposure can also affect cardiovascular responses to submaximal exercise. When shivering occurs during exercise in the cold, cardiac output is elevated relative to the cardiac output elicited by that same exercise intensity in temperate conditions. However, the increment in cardiac output simply reflects the requirement for increased systemic oxygen transport to sustain shivering, so cardiac output for any given oxygen uptake level remains unchanged (95,96).

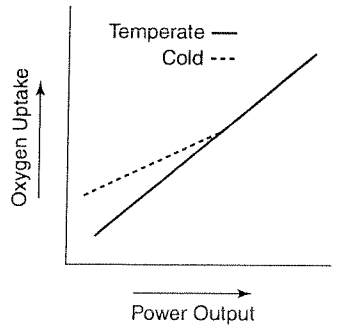

FIGURE 26.17 Steady-state oxygen uptake response during submaximal exercise at given power output in temperate and cold conditions. *(From Young AJ, Sawka MN, Pandolf KB. Physiology of cold exposure. In Marriott BM, Carlson SJ, eds. Nutritional needs in cold and in high-altitude environments. Washington: National Academy, 1996, 137.)*

Nevertheless, shivering during exercise could somewhat increase cardiovascular strain for a given submaximal exercise bout over the strain of that same bout without shivering. Cold exposure can also alter the way that a given cardiac output is achieved (95,96). During exercise in cold air or cold water, bradycardia and increased stroke volume are observed for a given cardiac output and oxygen uptake level compared to exercise at the same oxygen uptake in temperate conditions (95,96). Whether a baroreflex, a change in the inotropic state of the heart (cold-induced increase in plasma norepinephrine), or the effects of increased central blood volume resulting from peripheral vasoconstriction accounts for the bradycardia and stroke volume increase seen during exercise during cold exposure await definitive experimental determination (95,96).

Muscle Energy Metabolism

Cold exposure may also affect muscle energy metabolism during exercise. If shivering occurs during exercise, higher blood lactate concentrations may be attained in cold than in temperate conditions, but when oxygen uptake is the same (i.e., no shivering), blood lactate accumulation during exercise in cold and temperate conditions is the same (31). Muscle glycogen depletion has been observed to be more pronounced during low-intensity (e.g. below 25% maximal oxygen uptake) exercise in cold, when oxygen uptake levels were elevated compared to an exercise bout similar in intensity and duration performed under temperate conditions, but differences are not seen when bouts of higher-intensity exercise are compared (31). Earlier interpretations of that observation were that added energy requirements of shivering increased glycogen breakdown during exercise and that shivering preferentially metabolized muscle glycogen as an energy source. However, recent findings show instead that shivering, like low-intensity exercise, relies on lipid as the predominant metabolic substrate, but blood glucose, muscle glycogen, and even protein are metabolized as well (97). The accelerated muscle glycogen utilization previously observed during low-intensity exercise in cold conditions might result from increased motor unit recruitment to compensate for impaired force generation induced by muscle cooling. However, cold exposure would probably have to be extreme or entail prolonged immersion in cold water to induce a sufficiently pronounced decline in muscle temperature to impair force generation during sustained dynamic exercise (92).

Body Fluid Balance

Cold exposure is thought to influence both physiological and behavioral mechanisms affecting body fluid balance during exercise. For one thing, fluid intake can become inadequate during cold exposure. Thirst sensation may be blunted in cold persons, and cold environments often present practical constraints on voluntary drinking (frozen drinking supplies, desire to avoid urinating outdoors in the cold). Cold exposure is also thought to exacerbate fluid losses, but the extent that these effects are operational during exercise is unclear.

Perhaps the most widely recognized effect of exposure to cold air or immersion in cold water is an increased urine flow rate, or cold-induced diuresis, but controversy still exists regarding its precise mechanism. Experimental findings appear to rule out hormonal mechanisms and suggest that the increased systemic and renal blood pressure associated with cold-induced vasoconstriction may increase filtration and decrease reabsorption of water and solute by the kidney (98). However, whatever the mechanism or mechanisms, exercise counteracts or blocks them, because cold-induced diuresis is prevented by even moderate exercise during cold exposure (98).

Breathing cold air has also been suggested to exacerbate respiratory water loss during exercise, since cold air has lower water content than warmer air, and it is assumed that each inspired breath warms to body temperature and becomes 100% saturated with water as it passes through the respiratory passages (98). However, that assumption may not be valid. Experimental observations (99) illustrate that cold inspired air (−12°C) does not warm to the same temperature as warmer air (23°C), and in fact, less rather than more water was lost in the exhaled cold air. The difference in temperature between ambient and inspired air temperature becomes even greater when ventilation rate increases, as during exercise (99). Therefore, respiratory water losses are probably no more important for fluid balance during exercise in the cold than in warm conditions, and the most significant avenue of fluid loss during exercise in cold is the same as in warm conditions—sweating.

Even in cold environments, metabolic heat production can exceed heat loss, with the resulting heat storage causing hyperthermia and initiating thermoregulatory sweating. The problem is that clothing insulation needed for warmth and comfort in cold environments is much higher during rest and light activity than during strenuous activity (5). Therefore, if one begins exercising vigorously while wearing clothing selected for sedentary activities in the cold, sweating and the resultant drinking requirements can increase substantially. If those increased drinking requirements go unmet, dehydration will ensue, just as with exercise in the heat, with possibly similar adverse physiological and performance consequences (100) as discussed earlier in this chapter. Otherwise, there is little evidence that cold exposure has any unique impact on regulation (101).

Exercise Performance Limitations

As discussed earlier, cold exposure can reduce maximal aerobic power and increase metabolic and cardiovascular strain during submaximal exercise, both of which would be predicted to limit or impair exercise performance to some extent. However, as described, both of those effects appear to be mediated by significant body heat loss and cooling of deep

body and skeletal muscle. Thus, how exercise influences the ability to maintain thermal balance in the cold is probably the key factor determining whether cold exposure is associated with performance decrements. In addition, certain pathophysiological responses elicited by cold exposure can degrade performance by impairing an individual's ability to exercise.

Exercise and Thermal Balance

As described earlier, exercise elicits both thermogenesis and an increased peripheral blood flow (skin and muscles), which facilitates body heat loss by enhancing convective heat transfer from the central core to peripheral shell, and both responses increase concomitantly as exercise intensity increases. During exposure to cold air, the thermogenic response during exercise is sufficiently pronounced that the increased metabolic heat production usually matches or exceeds any exercise-related facilitation of heat loss, and exercise performance would not be affected by inability to maintain thermal balance (3). In contrast, because the higher convective heat transfer coefficient is so much greater during water immersion than in air, the exercise-associated increase in heat loss during exercise in water can be so large that metabolic heat production during even intense exercise is insufficient to maintain thermal balance (3).

Exercise affects thermal balance during cold exposure in two other ways. Arms have a greater ratio of surface area to mass and thinner subcutaneous fat than legs, so exercise-induced increments in muscle and skin blood flow to the limb tend to increase convective heat transfer more during arm than leg exercise (14). As a result, metabolic heat production is less effective for sustaining thermal balance during arm than leg exercise at a similar intensity (3). In addition, limb movement disrupts the stationary boundary layer of air or water that develops at the skin surface in a still environment, and the loss of that insulation further favors increased convective heat loss from the body surface. Experimental observations confirm that these effects have a measurable influence on thermal balance during exercise in cold water (102). The practical implications of those effects for performance in the cold remains to be fully explored, but they may have implications for design of cold-weather athletic clothing.

Hiker's Hypothermia: Exertional Fatigue and Thermoregulation in the Cold

Strenuous exercise can lead to exertional fatigue, and when strenuous exercise and high levels of energy expenditure are sustained for long periods, people have difficulty maintaining sufficiently high energy intake to offset expenditure. Fatigue due to exertion, sleep restriction, and underfeeding impairs an individual's ability to maintain thermal balance in the cold (103), and an anecdotal association between exertional fatigue and susceptibility to hypothermia has also been reported (104). This syndrome has been referred to as hiker's hypothermia. The simplest explanation proposed is that prolonged exercise produces fatigue, so the exercise intensity and rate of metabolic heat production that can be sustained decline, and if ambient conditions are sufficiently cold, negative thermal balance and declining core temperature ensue. When underfeeding is a factor, effects probably result from development of hypoglycemia, since acute hypoglycemia impairs shivering through a central nervous system–mediated effect. Also, declining peripheral carbohydrate stores probably contribute to an inability to sustain exercise or activity and the associated exercise-induced thermogenesis in the cold. Recent studies also indicate that shivering and peripheral vasoconstriction may themselves be directly impaired following strenuous exercise (75,105), repeated or prolonged cold exposure (106), or both. Irrespective of mechanism, when strenuous exercise produces fatigue, either acute or chronic, ability to maintain thermal balance appears to be degraded, and that in turn can further limit performance.

Cold-Induced Bronchospasm

For the most part, inhaling cold air during exercise has negligible effects. Upper airway temperatures, which normally remain unchanged during exercise under temperate conditions, can decrease substantially when extremely cold air is breathed during strenuous exercise, but temperatures of the lower respiratory tract and deep body temperatures are unaffected (107). Pulmonary function during exercise in healthy athletes is usually unaffected by breathing cold air, but allergy-prone athletes frequently have bronchospasm (108). The triggering mechanism for bronchospasm in susceptible persons remains unresolved, but it may be related to thermally induced leakage in the lung's microcirculation with subsequent edema (99). Persons who have bronchospasm when breathing cold air during heavy exercise exhibit a reduced forced expiratory volume (108), which can limit maximal ventilation, thus maximal performance. Further, even healthy persons can have an increase in respiratory passage secretions and decreased mucociliary clearance when breathing very cold air during exercise, and any associated airway congestion may impair pulmonary mechanics and ventilation during exercise, also impairing performance (109).

SUMMARY

Body temperature is regulated by two distinct control systems, behavioral and physiological. Behavioral thermoregulation involves the conscious, willed use of whatever means are available to minimize thermal discomfort. Physiological thermoregulation employs unconscious responses that are controlled by the autonomic nervous system in proportion to the core and skin temperatures. The physiological responses modifying body heat loss are increased skin blood flow and sweating, and those modifying heat conservation are vasoconstriction and shivering.

A MILESTONE OF DISCOVERY

Does central fatigue contribute to reduced exercise performance associated with hyperthermia? Human tissues retain their function and tolerate temperatures above 40°C without injury, but irrevocable damage can result beyond tissue temperatures above 42°C (1,110). Therefore, an inability or unwillingness to exercise beyond destructive internal body temperatures may be a type of protection against catastrophic hyperthermia. Animal research has shown that exercise performance can sometimes decrease when hypothalamic temperature exceeds 42°C (independent of body temperature), suggesting that very high brain temperatures can mediate protective thermoregulatory behavior (111). However, such high brain temperatures would not normally be expected in exercising humans.

Prior to the study of Nybo and Nielsen (42) there was no conclusive evidence that central fatigue contributes to reductions in exercise performance in the heat for humans. These investigators had 14 subjects exercise at 60% of $\dot{V}O_{2max}$ for 1 hour in a hot (40°C) and temperate (18°C) climate. Immediately after that exercise bout, subjects performed 2 minutes of sustained MVC of handgrip and knee extension with electrical stimulation (EL) periodically applied to the nervus femoralis. EL superimposed during the MVC generated the maximal total force (MVC + EL) and maximal voluntary activation percentage (MVC / MVC + EL). Core temperature increased to 40°C (exhaustion after 50 minutes of exercise) and 38°C (completed 1 hour of exercise) in the hot and temperate climates, respectively. Knee extension MVC force was initially unaffected by prior exercise, but after 5 seconds the decline in MVC force was greater after the hyperthermia than after the temperate trials. Total maximal force was similar, so the voluntary activation percentage was lower after exercise in hot (54%) than temperate (82%) conditions. Two subjects were passively heated to 39°C and demonstrated the same pattern of voluntary force decline during sustained maximal handgrip contractions. The experiments showed that hyperthermia significantly reduced force-generating capacity during a sustained MVC in both active (leg) and inactive (forearm) muscles compared to that measured at a lower core temperature. However, the capacity of the skeletal muscles was not altered by hyperthermia when EL was applied.

The study provides strong support for the idea that central fatigue may contribute to reduced capabilities for dynamic exercise in hot weather. It is unclear whether brain or other tissue temperature (or feedback inhibition from hot muscles) might have been influenced by the hyperthermia to send signals affecting motor activity. The mechanism for attenuated neuromuscular activity at a higher body temperature remains unresolved but may possibly be linked to the neurotransmitter serotonin within the brain, as discussed in the chapter.

Nybo L, Nielsen B. Hyperthermia and central fatigue during prolonged exercise. J Appl Physiol 2001;91:1055–1060.

Climatic heat stress and exercise interact synergistically and may push physiological systems to their limits. Heat stress reduces an athlete's ability to exercise. Physiological mechanisms contributing to this reduced performance include cardiovascular, central nervous system, and metabolic perturbations. Heat acclimatization provides many physiological adaptations that enable sustained exercise and protection from heat injury. Athletes routinely have high sweating rates during exercise heat stress that can lead to dehydration and adversely influence exercise performance.

In the cold, heat balance and requirements for shivering depend on the severity of climatic cold stress, effectiveness of vasoconstriction, and intensity and mode of exercise. Cold-induced vasoconstriction decreases blood flow to peripheral tissues, allowing them to cool and making them susceptible to cold injury. Cooling of peripheral tissues can degrade finger dexterity and impair skeletal muscle contractile function, while reduced core temperature can degrade the ability to achieve maximal metabolic rates and submaximal endurance performance. Body composition is the most important physiological determinant of thermoregulatory tolerance to cold exposure. The clothing requirement for warmth and comfort is much higher during rest and light activity than during strenuous activity, and overinsulation can cause heat stress that elicits sweating, wet clothing, and dehydration. Each of those factors can have undesirable affects on athletic performance and susceptibility to cold injury.

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